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OVERLAYING OF INFANTS.

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BY overlaying, strictly speaking, we mean suffocation of a child in bed due to some other sleeping person lying on him. The term is sometimes more loosely used to include suffocation of the child, for example, by bed-clothes, whilst in bed with another person.

Overlaying is said to be more common where there is overcrowding in poorer districts and as the result of the drunken habits of some parents. Professor Sydney Smith, in his text-book of forensic medicine, says that accidental overlaying of children causes quite an appreciable annual loss of life, and that overlaying is the most common form of accidental smothering.

Our experience suggests that very great care is necessary in these cases lest mistakes should be made.

In Melbourne in the last four years, March, 1948, to March, 1952, the bodies of 170 infants were brought to the City Morgue for examination as they had died suddenly or unexpectedly. Eleven of these infants were found dead in a bed in which one or both parents were sleeping at the time. In one case the dead baby was occupying the bed with his mother and another child.

In the absence of a post-mortem examination it could have been concluded that each of these deaths was due to overlaying. In fact, in each instance the body was brought to the morgue by the police with the suggestion that the child had been suffocated, and in Case VI described below the father came to the morgue himself in a most distressed state, crying, and said that he thought he must have lain on his child during the night.

Post-mortem examination was ordered by the coroner in each instance. The findings and circumstances were as follows.

CASE I.—A female child, aged two weeks, was dead in bed beside her mother when the mother woke up in the morning. The child had been born with bronchitis and jaundice. She had had a cough and nasal discharge for some days prior to death. There was a developmental defect in front of the left ear; the *foramen ovale* and *ductus arteriosus* were not closed. There was left *otitis media*. The lungs microscopically were found to contain patchy collapse and pneumonia. There was extra-medullary hæmatopoiesis in the liver.

CASE II.—A female child, aged ten weeks, died in a double bed with her parents. The mother was wakened by the baby making gasping noises at death. The child weighed four pounds five ounces, and there was gross congenital heart disease. Three hours before death the child's crying had wakened the parents.

CASE III.—A male child, aged five months, was dead when the parents woke up in the morning. He was lying on his back not covered by bedclothes. Examination showed bronchopneumonia. The child had been subject to "colds" since birth.

CASE IV.—A male child, aged nineteen months, was dead in bed beside the mother when she woke up in the morning. He had been restless, and the mother thought that he had been ill for twenty-four hours before death. Post-mortem examination showed acute pericarditis, acute pleurisy and acute bronchitis. *Neisseria meningitidis* was grown from the pericardial fluid.

CASE V.—A female child, aged three weeks, was dead in bed with the mother and another child. The mother had taken the child into her bed during the night because she was crying. A picture of an asphyxial mode of death was found macroscopically at autopsy, and microscopically there was acute bronchitis.

CASE VI.—A female child, aged two and a half years, was in a double bed with her parents. She was dead lying between them. She had been recovering from chicken-pox. She had had a running nose for two days before death. There were chicken-pox scabs on the skin. The trachea and bronchi were red and injected; microscopically tracheo-bronchitis was present.

CASE VII.—A female child, aged nine months, was dead in bed with her parents, who stated that she had suffered from gastro-enteritis for one week prior to death. The body was somewhat dehydrated. The liver was very pale and fatty, and microscopically there was gross fatty change.

CASE VIII.—A female child, aged eleven weeks, was dead in bed with the parents. Autopsy revealed laryngo-tracheo-bronchitis.

CASE IX.—A female child, aged ten days, was dead in bed with the mother. Autopsy showed bilateral *otitis media*.

CASE X.—A male child, aged fifteen months, was dead in bed with the mother. He was known to have had a temperature of 101° F. the night before death, and had vomited at 11 p.m. He had been given a "Sulphex" tablet. He was alive and apparently well at 6 a.m., but was dead at 7.30 a.m. Autopsy showed a blotchy skin rash, and meningococcal septicæmia.

CASE XI.—A male child, aged eleven weeks, was dead in bed between the parents. He was not covered by bedclothes. The father stated that the child had been put to bed apparently well. The parents had gone to a party and had come home sober about 3 a.m. The only finding at autopsy was an obvious increase in straw-coloured pericardial fluid. No specific cause of death was found.[1]

In the first ten cases it was decided that death was due to natural disease. In Case XI the only abnormal finding was a noticeable increase in pericardial fluid; no other definite pathological lesion was found. The autopsy was not as complete as it could have been as it was not pursued beyond the macroscopic and microscopic stage. At the same time it should be noted that the usual so-called asphyxial picture was not found, so that if one ignores the finding of increased pericardial fluid, the most that could be said by a forensic pathologist would be that death was consistent with overlaying. I questioned the father carefully about this child's death, but could come to no satisfactory conclusion. The father said that when he woke up in the morning the baby was lying dead on his back between the father and mother and his face was clear of the bedclothes. It would seem therefore that in Melbourne in the past four years there has been one possible case of overlaying.

In the absence of a careful autopsy it should never be concluded that a baby has been overlain simply because he is found dead in bed with his parents.

Pathologists who routinely carry out post-mortem examinations at the request of the coroner know how misleading circumstantial evidence about a death can be. One could quote numerous examples in which death was proved by autopsy to be due to natural causes, when, judging on the circumstances alone, the death was at first thought to be due to accident or murder.

What could be more natural than a mother taking a crying child out of his cot into bed with her during the night to comfort him and to allay the disturbance he is making?

At the Queen Victoria Hospital, Melbourne, 108 mothers attending the out-patient department were asked this question: "Do you ever take your baby into bed with you if he cries in the night?" Thirty-two said "Yes" and 76 answered "No". The same question was put to some mothers by a general practitioner with a large middle class suburban practice, and just over half of the women said that they took the baby into bed with them at night, especially if he was sick and so that father could get his rest. In a good residential outer suburb a sister in charge of a health centre asked 26 mothers if they ever took the baby into bed with them at night. Thirteen of them said they never did this; the remainder did it on occasions; four of them said that they did it only to feed the child.

A parent may not realize that the crying child she is taking into bed with her at night may be acutely ill; and should that child happen to die in his parent's bed from natural disease, the danger is that death from natural causes, in the absence of careful investigation, may be attributed to overlaying.

In June of this year the body of a male child, aged thirteen weeks, was brought to the City Morgue. It was alleged by the police to have been overlain by the mother. It was said that the mother and father had returned home late one evening and that the mother took the child to bed with her to breast feed him. The mother fed the child and then allowed him to go to sleep on the outer side of the bed, the child sleeping with his head lying on the elbow of the mother's outstretched arm. When the father woke up at six o'clock the following morning, the baby was dead. He was lying on his back, his face and head clear of any bedding and clear of the mother's body, with the back of his head still lying across the mother's elbow. The mother was accustomed to sleeping on her back.

Post-mortem examination showed that the body was that of a well-nourished, well-developed child. There were what appeared to be a finger-nail scratch mark on the front of the nose, a minute abrasion on the prominence of the right cheek, and four minute abrasions on the left side of the upper lip. The body was pale. The larynx was red. The trachea was injected. There was some very thin mucopus in the left middle ear. The blood was dark and fluid. There were no petechial hæmorrhages. There was a quantity of frothy fluid in the main bronchi and some curdled milk had been inhaled into the bronchial tree. The stomach contained curdled milk.

Microscopic examination showed laryngo-tracheo-bronchitis. The appearances in one of the larger bronchi are illustrated in Figure I.

It was the mother's habit always to take the baby into bed with her at night. She had two other children, aged six years and four years. She had followed this procedure with each of her babies. When she was questioned, she stated this quite frankly and said that as it had always been her procedure she did not believe that the deceased baby had died by overlaying. If she had overlain the child she would reproach herself for the rest of her life. Further inquiries elicited the fact that the deceased baby had suffered from a "cold" for two days prior to death, and the parents had had the child out visiting during the afternoon and evening of his death. The mother had bought some "Argyrol" and had put this into the child's nostrils for two days prior to death. The mother also stated that the baby had been very vigorous, and that he had frequently scratched his face —she had paid particular attention to cutting his nails in view of this. She said that on the day preceding his death he had scratched his face and made the marks previously described. It was at first thought that the marks on the child's face could have been made by buttons or other objects on the mother's clothing if she had accidentally lain on the baby, or that possibly he could have been wilfully smothered by a hand over the face. The inquiry and autopsy in this particular case showed that death was due to natural causes.

The diagnosis of overlaying is a serious one for the parent concerned, for at the inquest the coroner could bring in a finding of death by accident or misadventure or manslaughter or murder. Here is an interesting example.

In Melbourne, in 1897, a mother was committed for trial on a charge of manslaughter. The charge arose out of a coronial finding of overlaying. The mother was said to have arrived home under the influence of alcohol and taken her eleven months old son to bed with her. When she awoke the child was dead, and she thought that she had overlain him. A post-mortem examination was made which consisted of macroscopic inspection of the organs. The pathologist reported that there were a few petechiæ on the surfaces of the lungs, that there was some frothy fluid on sectioning the lungs, and that the blood was dark and fluid throughout the body. Nothing else of note was found apart from enlargement of the mesenteric lymph glands and prominence of the lymph follicles in the intestine. On this evidence it was concluded that death was due to overlaying.

At the trial the judge directed the jury to the effect that if they believed the allegations against the woman they could

[1] The heart weighed 45 grammes; this, with the pericardial fluid, could account for death on natural grounds.

ILLUSTRATIONS TO THE ARTICLE BY K. M. BOWDEN.

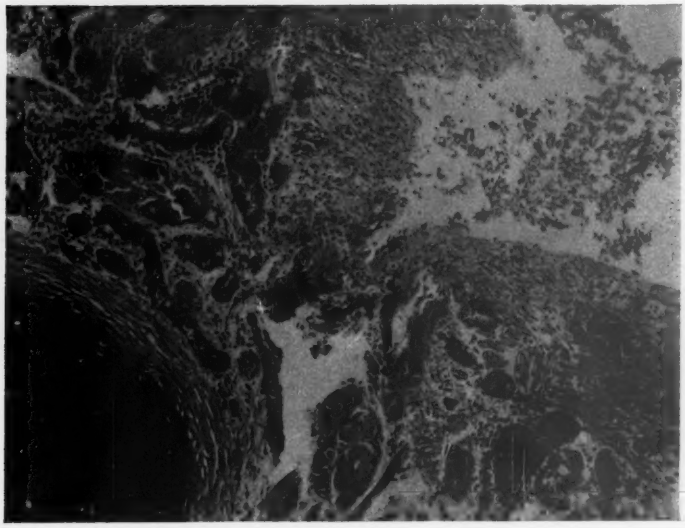


FIGURE I.
Photomicrograph of bronchus referred to in the text showing destruction of epithelium and infiltration of mucosa with cells.

ILLUSTRATIONS TO THE ARTICLE BY M. C. MOORE.

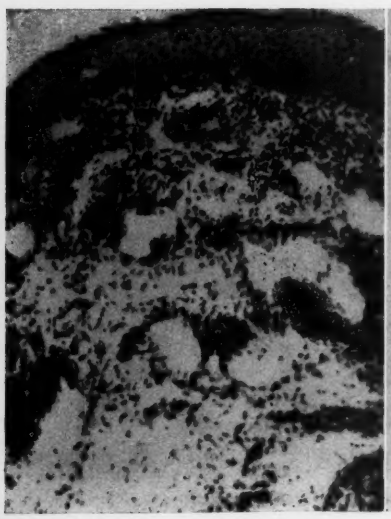
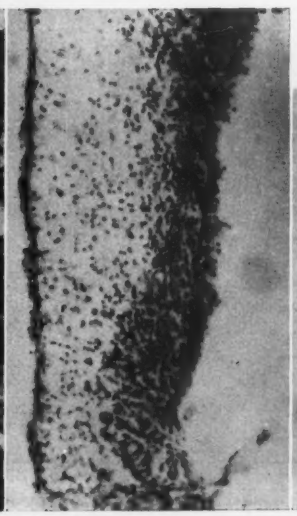


FIGURE III.



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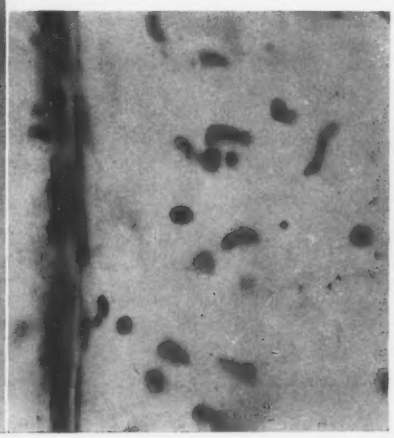
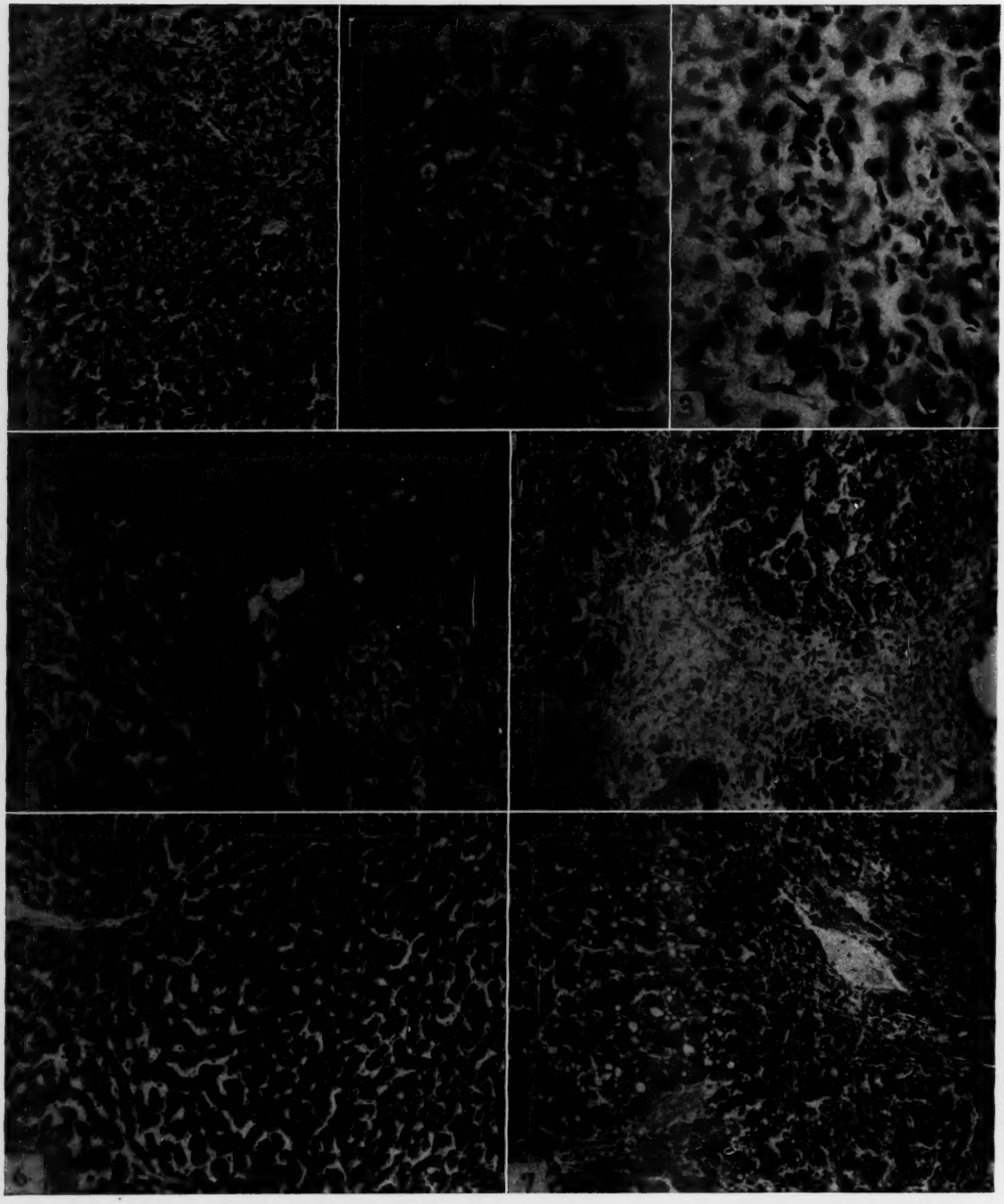


FIGURE V.

ILLUSTRATIONS TO THE ARTICLE BY ERIC G. SAINT.



ILLUSTRATIONS TO THE ARTICLE BY THOMAS F. ROSE AND EVA SHIPTON.

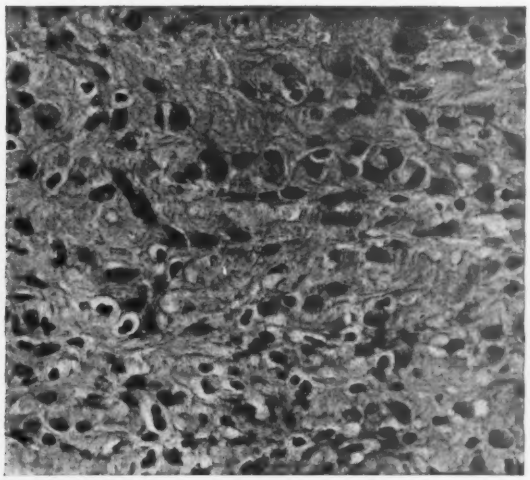


FIGURE I.

ILLUSTRATIONS TO THE ARTICLE BY R. A. MONEY.

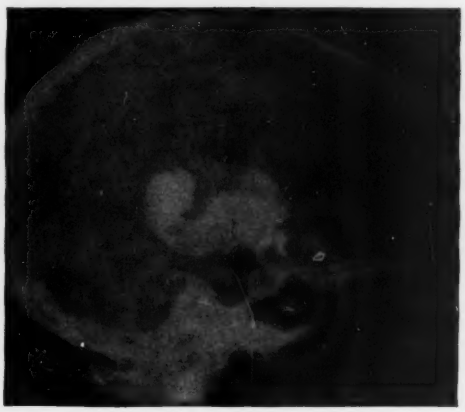


FIGURE I.

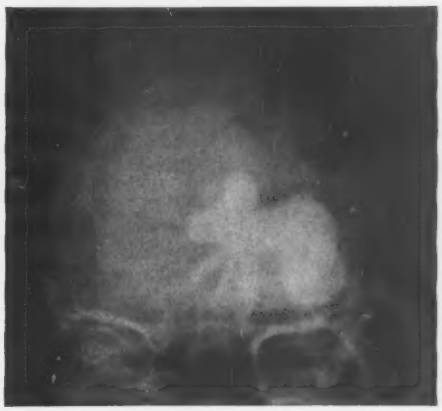
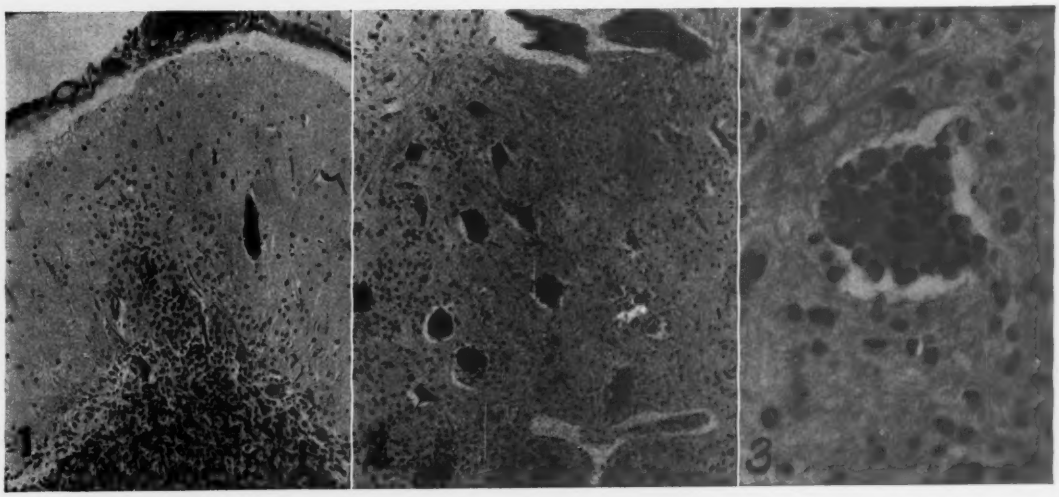


FIGURE II.

ILLUSTRATIONS TO THE ARTICLE BY A. K. GARVEN, J. MARGOLIS AND E. L. FRENCH.



ILLUSTRATIONS TO THE ARTICLE BY B. B. BARRACK AND R. E. POWELL.

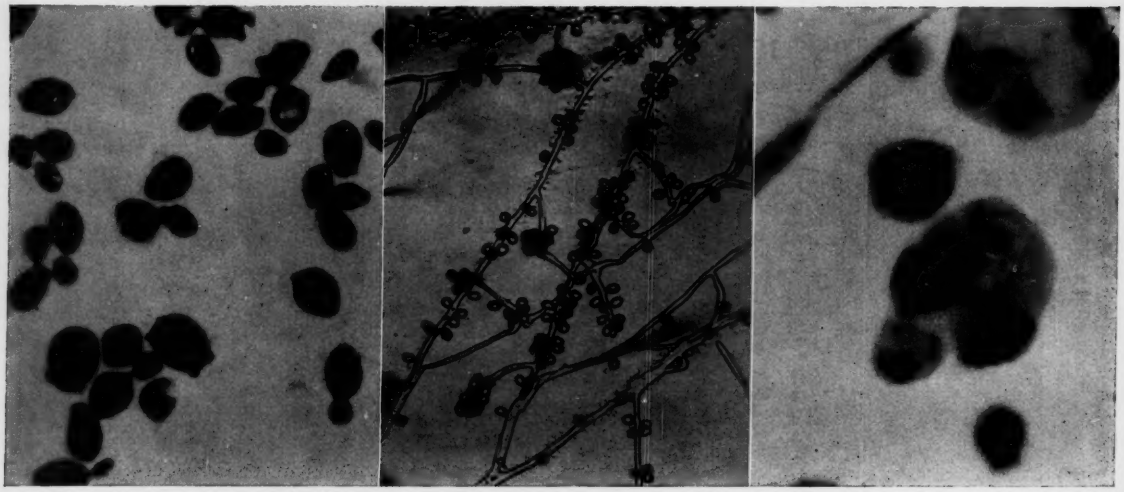
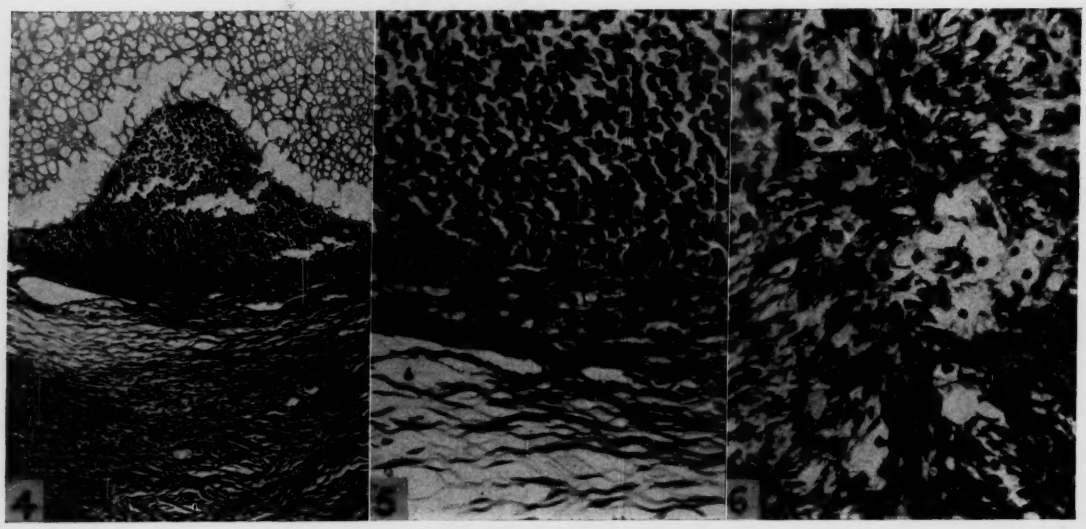


FIGURE II. FIGURE III. FIGURE IV.

ILLUSTRATIONS TO THE ARTICLE BY RICHARD FLYNN.



ILLUSTRATIONS TO THE ARTICLE BY R. T. W. REID.

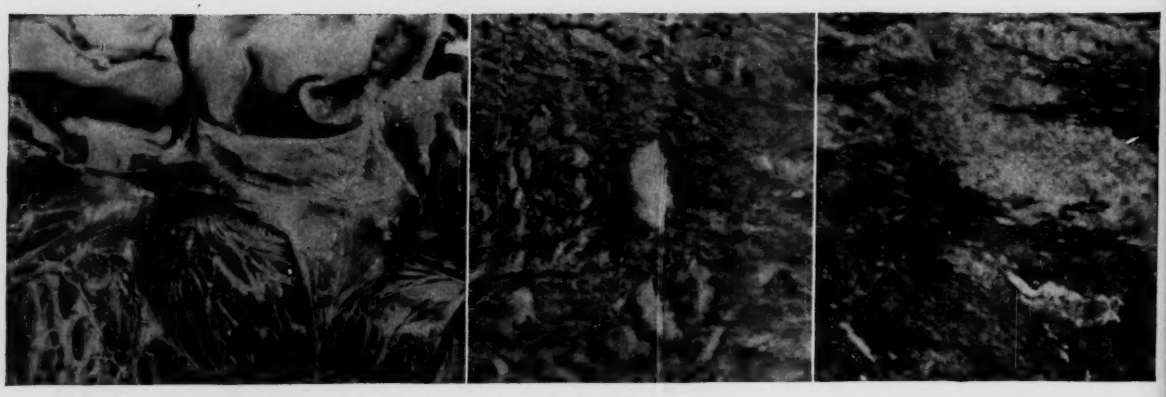


FIGURE I. FIGURE II. FIGURE III.

bring in a finding of manslaughter. The woman was found guilty of manslaughter, but before proceeding further the judge asked for the opinion of the State Full Court with regard to his direction to the jury. The Full Court delivered its judgment: "We think the proposition involved in this case is too broad and that, looking at all the circumstances, the charge of manslaughter cannot be supported. If a woman has made a resolution to kill her child, and having allowed herself to become in some degree drunk, takes it to bed with her knowing that in a heavy sleep she will probably overlie the child—apparently innocently, but at the same time with the intention to destroy the child—then that is murder. If, being in the state I have mentioned she knowing that she may overlie the child, and against the advice or disregarding the remonstrances of her friends, takes the child to bed with her and overlies it, killing it, that is manslaughter. But the evidence in this case is to the effect that the defendant had been drinking, and while under the influence of liquor and after taking the child to bed with her, by an unhappy mischance overlay it; this in our opinion, is not sufficient to support a charge of manslaughter." The woman was discharged.

In *The Lancet* in 1950 there was a report of the Ministry of Health for the year ended March 31, 1949. The section on child health in this report refers to the increase in deaths of infants under one year of age ascribed to accidental mechanical suffocation. Through the maternity and child welfare group of the Society of Medical Officers of Health, information was collected in 94 cases. In 49 of these 94 cases, death was ascribed to inhalation of stomach contents, in 42 to smothering, and in only three to disease —that is, tracheo-bronchitis or pneumonia. In 68% of the cases the maternal care was regarded as good or satisfactory. The report goes on to state: "It is still common for no autopsy to be made on a case of alleged mechanical suffocation, reliance apparently being placed entirely on circumstantial evidence at the inquest." In this series of 94 cases there were 42 in which death was recorded as due to smothering in the infant's cot or in his parent's bed, but autopsies had been carried out in only 24 of these, and, states, the report, "it appears still more uncommon for any microscopic examination to be made, the findings of inhaled stomach contents or the signs of asphyxia being accepted as the cause of death rather than as possibly merely terminal events . . . the cooperation of coroners and pathologists is needed to elucidate the causes of the increased incidence of these deaths".

No coroner should bring in a finding of overlaying on circumstantial evidence alone, no matter how strong, nor in such a case should he accept the pathologist's opinion of asphyxial death unless a very careful autopsy has been conducted.

Overlaying of an infant may be a deliberate act, but apart from that it should never take place. On no account should parents take a baby into bed to sleep with them. The risk of overlaying can be eliminated by placing the baby or child to sleep by himself, even if it is only on the floor.

It should be emphasized that a baby may die suddenly or unexpectedly from natural disease, and when that happens he may be in bed with his parents.

How many cases of alleged "overlaying" in the past have really been deaths from unsuspected natural disease?

SOME OBSERVATIONS ON THE USE OF THE CUTLER UNIVERSAL INTEGRATED OCULAR IMPLANT.

By M. C. MOORE,
Adelaide.

THE fixed staring appearance of an artificial eye has long been accepted as inevitable, though some small degree of movement has been obtained by some surgeons by the use of a glass ball buried within the socket. In recent years considerable ingenuity has been demonstrated, particularly by American surgeons, in developing by the use of an ocular implant a means of producing movement in the prosthesis which may rival that of the normal eye. The very important minor excursions noticed in the eyes of a person during conversation may be imitated so that one does not readily detect that one eye is artificial. The implant also largely prevents the sunken appearance of the eye and upper lid.

Many different types of implant are in use. Most are composed of acrylic, tantalum or combinations of these. The principle of their use is common to all. The implant is placed within Tenon's capsule and the muscles are attached in such a way that the ball moves in a similar manner to the eyeball. This movement is then transmitted to the prosthesis. In the "integrated" type the implant is incompletely buried and a peg on the back of the prosthesis fits into a hole on the bared anterior surface of the ball; in the magnetic buried type the prosthesis is held by the attraction of a permanent magnet.

The ocular implants used in the following series of cases were of the pattern described by Cutler in 1949. The majority were all-tantalum to avoid possible sensitivity to the plastic material contained in the earlier implants. They consist of a near-spherical ball, whose anterior surface is flattened and bears a hole to fit the peg of the prosthesis. Adjacent to this surface is a collar of tantalum meshwork, to which the muscles are sutured and through which the tissue grows to become very firmly attached. Several different techniques of insertion were used. Similar ones have been described by Cutler (1949) and recently by John Bignell (1952).

The purpose of this article is to discuss the techniques used and in particular the difficulties encountered. It is important to consider the latter lest it should be imagined that the subject is a closed one and that the results are uniformly successful. The complications are many, even to extrusion of the implant. The surgeon and the patient should realize that the method is still largely experimental, that failures are not uncommon and that careful treatment and long-lasting hygiene of the socket must be practised to gain satisfactory results.

Techniques.

In the following series three patients were subjected to evisceration of the eye, nine to enucleation of the eye, two to enucleation followed by complete burial of the implant, and six to reimplantation. The follow-up period on these cases varies from three months to two and a half years.

Evisceration.

A technique for evisceration of the eye suggested by Dr. A. L. Tostevin was found to be simpler than that of Cutler, and its results were quite as good. Evisceration is carried out in the usual manner. A curved incision is then made through the conjunctiva, Tenon's capsule and the sclera. This extends from a site just below the lateral rectus muscle and five millimetres behind the limbus in a direction downwards and backwards to a point beneath the inferior rectus muscle at the equator, or beyond if necessary to allow insertion of the implant. This is introduced through the wound, and the sclera at the limbus is trimmed to fit accurately the raised platform on the front of the implant. If the apposition here is close, the only suturing necessary is the repair of the scleral and conjunctival wounds with catgut. The incisions are made just large enough for the introduction of the implant.

The movement obtained in the prosthesis following this procedure was particularly satisfying. Extrusion did not occur in any of the three cases in which this method was used in the last two years, and discharge from the conjunctival sac was slight or moderate.

Unfortunately in most cases in which removal of an eye was indicated, enucleation was required rather than evisceration.

Enucleation.

Enucleation was carried out in nine cases of this series, Cutler's technique being followed. The implant was placed in Tenon's capsule and the recti were sutured to the gauze collar. Conjunctiva and muscle were pulled well up so as to overlap the platform of the implant. It was found then that there was less tendency for the tissue to retract

leaving the tantalum meshwork in part bared. This retraction and the resultant discharge were the chief difficulties encountered with this procedure. In two cases the modification described by James R. Hudson (1950) was used, and the results appear promising. Cutler and others have found more recently that the edge of the conjunctiva should be turned in at the margin of the bare surface of the implant to prevent granulation and to seal off the space deep to the gauze. This procedure has been followed in the last three cases.

Burial of Implant.

In two children enucleation was performed and the implant buried beneath the conjunctiva after suture of the recti muscles to the gauze collar so that they too overlapped the platform on the front of the implant. This procedure, as in the use of a glass ball, prevented a sunken appearance of the prosthesis, produced better movement in the floor of the socket, and might be expected to give a more permanent result than a glass ball. It produced a much cleaner socket than the usual method of insertion of the implant, but movement was considerably restricted.

Reimplantation.

An attempt was made in six cases to insert an implant into a socket from which the eye had been removed several years previously. Here Cutler's technique was followed, but the results were most disappointing. Including the cases in which the recti could be separated and sutured to the gauze, only one implant remained in place nine months after its insertion. In four others the tissue shrank back, usually after recurrent conjunctival infections, until the implant was extruded. The remaining one was removed because of persistent discharge.

Difficulties Encountered in Use of the Implant.

Unfortunately it was found that persistent discharge and other less frequent complications marred the happy prospects of some patients fitted with an implant. One must therefore choose his patients from those who will be prepared to practise careful hygiene of the socket and who will accept the longer period of post-operative care necessary in order to gain the benefits a moving prosthesis offers. The complications have been discussed also by A. P. Drucker and other American oculists (Drucker *et alii*, 1941). The most important encountered here were extrusion of the implant and conjunctival discharge.

Extrusion of the Implant.

Extrusion of the implant was not seen after evisceration. Unfortunately, many cases occur in which possible supervention of sympathetic ophthalmia, diagnosis of intraocular tumour *et cetera* necessitate more complete removal of ocular tissues, and enucleation becomes the operation of choice. Of nine such cases extrusion occurred in three after periods of five to twenty-three months. The tissues gradually retracted from the gauze collar of the implant until a considerable area, usually situated above and laterally, was bared. This was associated with persistent discharge from the socket and displacement of the implant. In one case in which a second attempt was made to attach the tissues in the receding area, this was unsuccessful.

In the cases of "reimplantation" retraction occurred even though the implant was placed deeply behind a collar of Tenon's capsule. An attempt at undermining the tissue and resuturing when retraction had begun was unsuccessful in one case, and a second attempt at reimplantation with a new implant failed in another. That excellent movement of the prosthesis can be attained was shown in most cases before this complication arose.

Persistent Conjunctival Discharge.

Discharge from the socket in most cases was more than is usually found in a simple socket. It varied from a negligible amount to a quantity sufficient to necessitate cleansing several times daily. In over half the cases it was enough to cause patients to complain of inconvenience. It was worse in those cases in which the tissue had retracted back from the gauze. Culture of the exudate produced such varied organisms as *Pseudomonas pyocyanea*, *Staphylococcus albus* and coagulase-positive hæmolytic staphylococcus, and treatment with appropriate chemotherapeutic agents was disappointing. There seems to be a susceptibility to recurrent infection. Careful hygiene of the socket is essential, and "Salacrin" jelly used at night has been found useful. In spite of all measures one implant had to be removed to eradicate prolific discharge, from which on culture *Pseudomonas pyocyanea* was grown. This complication is the greatest cause of discomfort to the patient and concern to the surgeon.

Difficulty in Fitting a Comfortable Prosthesis with a Peg.

Apart from the technical problems met with in making a suitable prosthesis, for example, when the implant moves from the primary position, some patients complained of difficulty in inserting and particularly in removing the prosthesis. It was found that little movement was sacrificed by the use of a prosthesis without a peg provided that its concavity fitted approximately the platform on front of the implant, against which it is soon held by suction. Unfortunately the same result could not be attained if the front of the implant was completely covered by conjunctiva with complete burial of the implant. Much greater convenience can be gained with little loss of movement by discarding the peg of the prosthesis.

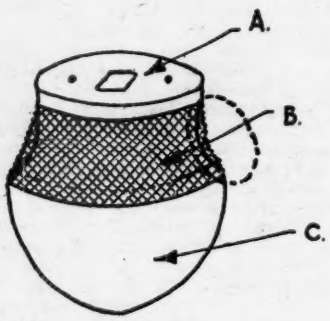


FIGURE I.

Diagram of the Cutler ocular implant. The area encircled is shown in detail in Figure II. A: Hole in anterior flattened surface to take the peg of the prosthesis. B: The collar of tantalum gauze. C: The smooth posteriorly placed surface.

Limitation of Movement.

The full range of movement of an eye is rarely attained in a prosthesis. Almost full excursions in the downward and lateral positions of gaze with adequate adduction and elevation are, however, the rule. Convergence is present in some cases, and I have had the pleasure of seeing in a moving film shown by Cutler a patient whose prosthesis imitated perfectly the nystagmus of the other eye. The results attained depend on the position of the implant as well as on its actual range of movement. There was a tendency in one case for the implant to become displaced outwards from the primary position over a period of some weeks with consequent limitation of lateral movement.

Histological Findings in the Tissue in Contact with the Implant.

In one case in which the implant had to be removed because of retraction of tissue in one sector, the tissue was shaved from both surfaces of the gauze in another healthy sector and submitted to histological examination. The regions from which sections were cut are indicated in Figure II. Figures III, IV and V are photomicrographs of these sections and show the histological features described

below. The tissue in contact with the gauze had the general characteristics of chronic granulation tissue with a considerable formation of fibrous tissue. An attempt was made to determine the fate of the epithelium at the free edge of the conjunctiva. Two alternatives suggest themselves: (i) the epithelium may undergo continuous necrosis and regeneration; (ii) it may turn down through the gauze and continue to grow backward in contact with the smooth surface of the tantalum separating the latter from the deep tissues (Figure II). It was found, in fact, that the

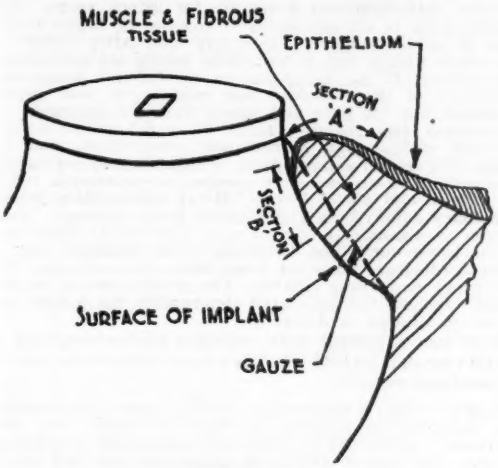


FIGURE II.
Guide to the areas from which tissue was taken in the preparation of the sections represented in Figures III, IV and V.

epithelium became continuous here with an "epithelium" of one to three layers of flattened cells arranged in contact with and parallel to the tantalum surface. The implant was retained by the growth through the tantalum meshwork of a fibrous granulation tissue which is very strong, removal of the implant requiring sharp dissection with scissors or knife. Behind the gauze the all-tantalum implant was unattached and the tissue in contact with it presented a smooth, shiny surface, but the tissue was firmly adherent to the acrylic here of an acrylic and tantalum implant.

Conclusions.

The Cutler integrated implant offers a means of ensuring a pleasant cosmetic result for a patient who must lose an eye. The sunken appearance of a socket and of the upper lid can be largely avoided and a degree of movement of the prosthesis gained which closely imitates that of the normal eye. Unfortunately, these advantages are often attained at the cost of some inconvenience from the exudate forming in the socket. This is avoided if a completely buried implant is used, and here the chance of extrusion is less. An implant similar to that of Cutler, but having its anterior surface completely covered by tantalum mesh, should be satisfactory. Such implants are already being used in the United States of America. The problem of transmitting movement from implant to prosthesis is then reintroduced. An ingenious method already used in the United States incorporates a permanent magnet in a buried implant in conjunction with a partly metallic prosthesis. It is to be hoped that some such method will prove successful in eradicating complications from a procedure which promises a great consolation to the patient faced not only with the loss of an eye, but with its substitution by a fixed, staring, obviously artificial eye in a somewhat sunken position.

Summary.

A series of cases is presented in which the Cutler universal integrated ocular implant was used after enucleation and evisceration of the globe. In the latter instance a new method of insertion is described. The results after evisceration were excellent, those after enucleation satisfactory. "Reimplantation" gave poor results, most implants being extruded later. The difficulties and complications encountered are discussed, and the histological findings in the tissue adjacent to the implant are described. Further work is necessary to overcome the complications encountered, especially extrusion and discharge from the socket, and it is suggested that some form of completely buried implant will prove most successful.

Acknowledgements.

I wish to express my appreciation to Dr. Malcolm Fowler and the staff of the Institute of Medical and Veterinary Science, for their assistance in preparing sections, and to Miss G. D. Walsh and Dr. A. D. Packer, for preparation of the photographs at the Medical School, University of Adelaide.

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Legends to Illustrations.

FIGURE III.—Section (marked A in Figure II) showing the conjunctival epithelium and tissue underlying it.

FIGURE IV.—Section (marked B in Figure II) showing the "epithelium on the left continuous with the conjunctival epithelium above and in contact with the smooth tantalum surface underlying the gauze.

FIGURE V.—High-power view of the "epithelium" of Figure IV.

INFECTIOUS HEPATITIS IN OLDER AGE GROUPS.

By ERIC G. SAINT,[1]

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AT the present time infectious hepatitis occurs sporadically in our community, and there is evidence that members of older age groups are among those who are infected. Since 1946 the Clinical Research Unit of the Walter and Eliza Hall Institute of Medical Research and the Royal Melbourne Hospital has shown continued interest in the problem of jaundice, particularly in infectious hepatitis. Of the 91 cases of infectious hepatitis studied, no less than 20 occurred in persons aged over forty years. It cannot be claimed that this age incidence is a true reflection of the state of affairs in the Victorian community as a whole, but it does indeed suggest that infection with the virus of infectious hepatitis is not entirely the prerogative of youth, as we have tended to suppose.

Apart from rare cases of acute hæmolysis, jaundice occurring in patients aged over forty years is traditionally regarded as a problem of surgical diagnosis; gall-stones, carcinoma of the pancreas or extrahepatic ducts, and secondary malignant deposits in the liver are the most frequent causes of obstructive jaundice. When the records of the 20 cases of infectious hepatitis in this age group were looked through, it was apparent that an original misdiagnosis was all too frequent, leading to laparotomy in several instances, with generally unsatisfactory results.

This study has therefore two objects in view—firstly to investigate in some detail the natural history of infectious hepatitis in this older age group, and secondly to examine

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the clinical, biochemical and histological features which differentiate jaundice of this type from that due to surgical obstruction.

Method of Study.

Twenty patients aged over forty years, suffering from infectious hepatitis, and referred to the Clinical Research Unit between 1947 and 1951 for investigation, have been studied in full clinical detail. The routine biochemical investigations carried out on each patient included a semi-quantitative estimation of urinary urobilinogen content, estimation of the serum bilirubin content, the Hangar cephalin flocculation test, fractionation of serum proteins, estimation of the serum alkaline phosphatase content and estimation of the prothrombin index. Other biochemical and radiological investigations were carried out when indicated. The majority of patients were also subjected to at least one aspiration liver biopsy by means of a standardized technique described previously by King and Perry (1948). The clinical, biochemical and other pertinent findings in this study are summarized in Table I. All patients have been comprehensively followed up in the out-patient department.

The sex of these patients was equally distributed (ten males and ten females). The mean age of the group was 55·0 years, the eldest patient being aged seventy-eight years. A history of contact with other subjects of infectious hepatitis was obtained in only four cases. In one case (Case 11), jaundice occurred about one hundred and twenty days after blood and plasma transfusion for second and third degree burns.

Reports of Cases.

Cases have been grouped arbitrarily, according to the severity or chronicity of the illness, into the following four groups.

Group I: Mild Icterus of Short Duration.

CASE 2.—O.P., a transport driver, aged forty-five years, was admitted to hospital on April 28, 1951, with a history of dull abdominal pain, nausea and vomiting followed a week later by jaundice. No history of contact was obtained. He had been a consistently heavy drinker for at least fifteen years. He was mildly jaundiced and the liver was slightly enlarged on palpation. No spider nævi were present. Liver function tests (see Table I) indicated probable infectious hepatitis. Examination of the liver biopsy material revealed necrosis and excessive infiltration of the lobules with macrophages and evidence of regeneration of parenchymal cells (see Figure I). His jaundice cleared rapidly. Persistent epigastric pain was a feature of convalescence, and a barium meal examination showed an irritable duodenal cap without a demonstrable crater.

Comment.—In the two cases in this "mild" group the prodromal symptoms were transient and the jaundice was slight and of short duration. The biochemical tests in one case gave equivocal results.

Group II: Moderate or Severe Icterus, No Complications or Sequelæ, a Fairly Prolonged Clinical Course.

CASE 3.—Mrs. V.K., a housewife, aged sixty-one years, was admitted to hospital on February 21, 1951. She was known to have been in contact with cases of infectious hepatitis, and five weeks previously she had complained of acute epigastric pain and nausea, followed by jaundice a week later. This had not cleared and was thought to be of obstructive type. She was obese, hypertensive and deeply jaundiced, and she had an enlarged liver. Biochemical tests (Table I) gave results typical of infectious hepatitis. Examination of the liver biopsy material revealed centrolobular necrosis and inflammatory cell invasion. The jaundice lasted two months, but eventually she made an uncomplicated recovery.

CASE 4.—Mrs. E.F., a housewife, aged forty years, underwent a cholecystectomy for gall-stones in 1946. She was admitted to hospital on May 24, 1949, after an illness which began suddenly with severe colicky epigastric pain and vomiting followed five days later by jaundice. She was thought to have recurrence of stones in the common bile duct. No history of contact was obtained. She was deeply jaundiced. The liver was demonstrably enlarged. Repeated vomiting required intravenous glucose therapy. A positive response to the cephalin flocculation test and a normal level of serum alkaline phosphatase pointed to a diagnosis of hepatitis rather than obstruction. Liver biopsy showed necrosis of liver cells with inflammatory cell invasion of the lobule typical of acute infectious hepatitis. She recovered after six weeks and has subsequently remained in good health.

The clinical history was suggestive of recurrent stones, but liver function test results were typical of infectious hepatitis.

CASE 8.—W.T., a male pensioner, aged seventy-one years, was admitted to hospital on January 17, 1950. He had been troubled with recurrent dyspepsia for fifteen years. Eight months prior to his admission to hospital he began to complain of severe epigastric pain one hour after meals. He lost much weight and a few weeks before his admission he was noticed to be jaundiced; a provisional diagnosis of carcinoma of the pancreas was made. He was severely jaundiced, but the liver and spleen were not enlarged. The biochemical test results (Table I) were compatible with hepatitis rather than extrahepatic obstruction, and liver biopsy (Figure II) showed many multinucleated regenerating liver cells, and an excessive number of wandering cells in the lobules and portal tracts. X-ray examination revealed pulmonary emphysema and calcified aortic plaques. Examination by means of a barium meal revealed an ulcer crater on the lesser curve and distortion of the duodenal cap. An electrocardiogram revealed total auriculo-ventricular block with rapid ventricular rhythm. The jaundice lasted for seven weeks. Despite the associated abnormality the patient made an uncomplicated recovery.

This was a patient with multiple gastro-intestinal and cardio-vascular lesions who developed infectious hepatitis of moderate severity.

CASE 10.—F.F., a male textile worker, aged forty-two years, was admitted to hospital on May 25, 1949. Six weeks previously he had felt feverish, had complained of a dull ache in the right upper abdominal quadrant and had vomited. Ten days later he was jaundiced. He had been in contact with a case of infectious hepatitis about six weeks prior to the onset of symptoms. The clinical history was suggestive of an infectious cause, but biochemical test results were more indicative of extrahepatic obstruction (Table I). Liver biopsy (Figure III) showed inconsiderable inflammatory cell invasion and many plugged bile capillaries. Two months after his admission to hospital the jaundice showed no sign of clearing and laparotomy was accordingly performed. No extrahepatic obstruction was noted. His post-operative course was uncomplicated and jaundice began to clear two weeks later.

This was a puzzling case of probable infectious hepatitis of the so-called cholangiolitic type, with prolonged jaundice, atypical results to biochemical tests, and histological findings more suggestive of obstruction.

Comment.—There were ten cases in this group. The prodromal symptoms were generally of acute onset and of considerable severity. Severe anorexia was accompanied by nausea and vomiting, and after four to ten days the appearance of bile pigments in the urine heralded the onset of frank jaundice. A point of practical clinical importance was the character and severity of the pain experienced. While many patients complained only of upper abdominal discomfort or of a dull ache beneath the right costal margin, in a significant number of cases the pain was intolerably severe, and no less than five patients in this group were admitted to hospital with a justifiable provisional diagnosis of jaundice due to stone in the common duct or carcinoma of the head of the pancreas. In all cases moderate enlargement of the liver was detected, and in six the spleen was palpable. The course of the disease was uncomplicated, but in comparison with the average case encountered in young people, unusually prolonged. Clinically obvious icterus persisted for a mean duration of eight weeks (range, five to twelve weeks). Biochemical tests were nearly always of help in differential diagnosis. In Case 10, however, in which there was a negative response to the flocculation test in association with elevation of the serum alkaline phosphatase content, there were several perplexing features, for the history was indicative of infection, yet biopsy revealed only a minimal degree of inflammatory reaction surrounding the small bile ducts. Similar cases of "cholangiolitic hepatitis" have been described by Watson and Hoffbauer (1946).

TABLE I.

Clinical, Biochemical and Histological Features in Twenty Cases of Infectious Hepatitis.

Group.	Case No.	Patient's Name, Age (Years), Sex.	Date.	Clinical Features.	Duration of Jaundice. (Weeks.)	Associated Disease or Condition.	Results of Biochemical Tests.[1]	Biopsy.	Course.
I: Mild.	1	E.K.; 59; F.	5.9.49	Painless jaundice; no enlargement of liver or spleen.	2	Coronary heart disease.	SB 2; CF nil; A 3·5; G 2·6; AP 14.	Slight fatty change.	Recovery.
	2	O.P.; 45; M.	28.4.51	Abdominal pain and jaundice. Liver slightly enlarged.	3	Chronic alcoholism.	SB 2; CF ++; A 3·7; G 2·2; AP 18.	Necrosis and inflammation.	Recovery.
II: Moderate.	3	V.K.; 61; F.	21.2.51	Pain and severe jaundice; slight enlargement of liver.	8	Essential hypertension.	SB 35; CF +++; A 4·5; G 2·5; AP 22.	Necrosis and inflammation.	Recovery.
	4	E.F.; 40; F.	24.5.49	Severe pain and jaundice. Liver enlarged 2 centimetres.	6	Previous cholecystectomy.	SB 60; CF +++; A 3·7; G 2·6; AP 12.	Necrosis and inflammation.	Recovery.
	5	A.F.; 41; M.	2.10.49	Painless jaundice with considerable enlargement of liver and spleen.	8	——	SB 17; CF +++; A 4·0; G 3·2; AP 33.	Active regeneration.	Recovery complicated by acute psychosis.
	6	E.S.; 46; F.	20.10.46	Abdominal pain and jaundice. Liver and spleen enlarged.	5	Malnutrition.	SB 20; CF nil; A 4·2; G 1·5; AP 17.	? Regeneration. ? Cholangitis.	Recovery.
	7	E.F.; 70; F.	15.4.51	Pain and jaundice. Enlarged liver.	6	Essential hypertension.	SB 25; CF +++; A 2·8; G 2·5; AP 15.	? Regeneration. ? Cholangitis.	Recovery.
	8	W.T.; 71; M.	17.1.50	Severe pain, weight loss and jaundice. No enlargement of liver or spleen.	7	Gastric and duodenal ulcers; coronary heart disease.	SB 20; CF +; A 4·1; G 2·2; AP 18.	Regeneration.	Recovery.
	9	F.E.; 47; M.	17.5.47	Painless jaundice. Enlarged liver and spleen.	9	Apical tuberculosis.	SB 33; CF +; A 4·3; G 2·2; AP 23.	Necrosis and inflammation.	Recovery.
	10	F.F.; 42; M.	29.5.49	Pain and jaundice. Enlarged liver and spleen.	12	——	SB 35; CF nil; A 4·1; G 2·3; AP 26.	? Extrahepatic obstruction.	Laparotomy; recovery.
	11	F.B.; 48; F.	22.6.51	Painless jaundice following blood transfusion. Liver and spleen enlarged.	10	Epilepsy; recent burns.	SB 20; CF +++; A 3·5; G 3·5; AP 47.	Necrosis and inflammation.	Slow recovery.
	12	H.H.; 61; F.	12.2.52	Painless jaundice.	5	Chronic pyelitis.	SB 30; CF +++; A 4·2; G 3·2; AP 27.	Inflammation and regeneration.	Recovery.
III: Severe.	13	A.S.; 52; F.	13.8.51	Painless jaundice; coma; diminished liver dulness; enlarged spleen.	3	Previous carcinoma of breast.	SB 60; CF +++; A 3·1; G 4·2; AP 19.	Massive necrosis.	Death in third week.
	14	A.F.; 40; M.	28.5.48	Pain and jaundice; enlarged liver and spleen; ascites; drowsiness.	12	——	SB 35; CF +++; A 2·9; G 1·1; AP 13; prothrombin 79%.	Late biopsies showed active regeneration and fibrosis.	Complicating septicæmia. Ultimate recovery with good liver function.
	15	A.D.; 48; M.	12.10.46	Severe pain and jaundice; drowsiness; enlarged liver and spleen.	3	Chronic alcoholism.	SB 35; CF +++; A 2·7; G 2·7; AP 30.	Massive necrosis.	Laparotomy; death in third week.
	16	A.G.; 57; M.	25.5.47	Painless jaundice; ascites; drowsiness. Enlarged liver and spleen.	10	——	SB 15; CF +++; A 3·03; G 3·75; AP 37; prothrombin 69%.	Late biopsies; regeneration and fibrosis.	Ultimate recovery with good liver function.
IV: Chronic.	17	E.H.; 47; F.	10.7.46	Four episodes of severe jaundice in 3 years.	8	——	SB 25; CF +++; A 4·1; G 2·7; AP 18.	Regeneration and fibrosis.	Late recovery with good liver function.
	18	M.S.; 51; F.	6.8.51	Chronic ill-health; ascites; melæna; enlarged spleen; coma.	—	Two pregnancies.	SB 2; CF ++; A 2·0; G 3·6; AP 20.	Massive fibrosis.	Death in liver failure following paracentesis and hæmorrhage.
	19	B.S.; 60; M.	17.12.50	Recurrent jaundice; enlargement of liver and spleen.	16	Essential hypertension and coronary heart disease.	SB 50; CF +++; A 3·2; G 4·5; AP 18.	Necrosis and regeneration with fibrosis.	Partial recovery with impaired liver function.
	20	McI.; 45; F.	10.7.49	Recurrent jaundice; ascites; melæna; splenomegaly.	—	Ulcerative colitis.	SB 9; CF +++; A 1·6; G 5·0; AP 10; prothrombin 30%.	Regeneration and fibrosis.	Chronic ill-health.

[1] SB, serum bilirubin (units per 100 millilitres); A, serum albumin (grammes per 100 millilitres); G, serum glubulin (grammes per 100 millilietrs); AP, alkaline phosphatase (King-Armstrong units per 100 millilitres); CF, cephalin flocculation.

Group III: A Fulminating Course ("acute yellow atrophy"), and Frequently a Fatal Termination.

CASE 13.—Mrs. A.S., aged fifty-two years, a housewife, was admitted to hospital in August, 1951. Two weeks before her admission she had complained of anorexia, nausea and vomiting. Jaundice developed a week later. On her admission to hospital she was deeply icteric. The liver was not palpated, but the spleen was just tipped. She was stuporose, but could be roused. Her breath had the characteristically sweet odour of liver failure. The left plantar response was extensor in type. Liver function tests revealed severe liver damage (Table I). She was treated with glucose (10%) and serum given intravenously, and a continuous intragastric drip was set up delivering five pints of milk daily. Two grammes of aureomycin were administered daily for seven days. Her condition appeared to improve temporarily, but she lapsed into coma again and died two weeks after her admission to hospital.

At autopsy numerous petechial hæmorrhages were found throughout the alimentary canal and the lungs. The liver was small and shrunken, and on section large yellow areas of necrotic tissue were present in both right and left lobes. Histologically there was widespread necrosis of parenchymal cells with a diffuse inflammatory reaction (see Figure IV).

This was a case of acute fatal fulminating infectious hepatitis.

CASE 14.—Mr. A.F., aged forty years, had an illness which had commenced gradually with fatigue, epigastric discomfort and jaundice. There was no history of contact. At the country hospital to which he was first admitted he lapsed into a state of incipient hepatic coma. He was transferred to the Royal Melbourne Hospital on May 28, 1948. He was deeply jaundiced. Both the liver and spleen were considerably enlarged and an erythematous rash was present on the arms and abdomen. Immediate biochemical investigations showed a strongly positive response to the cephalin flocculation test and low serum protein concentration (4·0 grammes *per centum*).

He was treated with glucose (10%), serum and protein hydrolysate given intravenously, and rallied from his comatose state. However, he developed an area of cellulitis at a transfusion site and a metastatic abscess developed in the right lung. Blood culture yielded a growth of a coagulase-positive *Staphylococcus aureus*. He was treated with penicillin and repeated transfusions of blood. After two weeks his temperature began to subside.

Liver biopsy was performed very late in the course of the disease and showed the picture of nodular hyperplasia (Figure V). He was discharged to convalescence in July, 1949, and a guarded prognosis was given. However, repeated biopsies showed a complete return to near-normal liver architecture (Figure VI), and he is now well and at work and has no demonstrable enlargement of liver or spleen.

This was a severe case of fulminating infectious hepatitis complicated by staphylococcal septicæmia. Despite gloomy prognostications recovery took place, with the gradual restoration of normal liver structure and function.

CASE 15.—Mr. A.D., a male alcoholic, aged forty-eight years, was admitted to hospital on October 12, 1948. Three weeks previously he had complained of severe upper abdominal pain which radiated to both shoulders, and of vomiting. He later became jaundiced. On examination of the patient the liver was considerably enlarged. In view of the severity of the pain the story was suggestive of extrahepatic obstruction; but biochemical test results indicated hepato-cellular jaundice.

He became increasingly drowsy. While doubt existed about the diagnosis laparotomy was felt to be justified, and his abdomen was explored under local anæsthesia, but no abnormality in the biliary tract was detected. He lapsed into coma and died in liver failure nine days after operation.

At autopsy the liver was found to be greatly shrunken. Microscopic examination revealed only a few islands of irregular regenerating liver cells in a mass of collagenous stroma, which contained thin-walled blood vessels, mononuclear cells and lymphocytes.

In this case the relative parts played by alcohol and infection were difficult to assess; but the acute onset of the jaundice suggested that the virus of infectious hepatitis may have been the major cause of his illness.

Comment.—There were four patients in this group of severe cases, two of whom died. After a protracted illness and convalescence the remaining two patients survived, and it is of interest that over an extended period of observation (four and five years respectively) it is apparent that their recovery is complete. The clinical features which indicate an infection of unusual severity are the depth of the jaundice, continued anorexia and persistent vomiting and a bleeding tendency. Drowsiness and mental confusion are the harbingers of hepatic coma. Abnormal plantar responses may be elicited at this stage.

Massive necrosis is suggested in the biochemical tests by low prothrombin levels (below 60%) and by significant lowering of the level of serum albumin in association with elevation of the globulin content, giving an albumin-globulin ratio of unity or less.

These patients were treated with glucose (10%), serum and protein hydrolysates ("Parenamine", Stearns) given intravenously, and by vitamins given parenterally. A reinforced milk mixture was delivered by the continued intragastric drip method through a Rehfuss tube (Garlick, 1948).

Any circumstances which impair the functional reserve of the liver render the organ more vulnerable in infection, and the two fatal cases illustrate the additional danger to life imposed by previous alcoholism and malnutrition, and by the stress of laparotomy.

Group IV: Chronic Hepatitis: A Clinical History of Relapses and Evidence of Permanently Impaired Liver Function.

CASE 19.—B.S., aged sixty years, a storeman, in January, 1950, felt tired and weak, and in March became jaundiced. This state continued for three months and was considered to be due to extrahepatic obstruction, but there was no confirmation at laparotomy. In August two-stage prostatectomy was performed for the relief of urinary symptoms. In September his jaundice recurred and it persisted until his admission to the Royal Melbourne Hospital on December 17, 1950.

He was moderately jaundiced and severely wasted, and had considerable hepatomegaly and an enlarged spleen. Mild arterial hypertension was present. The biochemical tests revealed grossly impaired liver function (see Table I), and the liver biopsy showed considerable liver damage typical of chronic infectious hepatitis (Figure VII). He was treated with penicillin and streptomycin with apparent improvement, and was discharged from hospital on January 30, 1951. In April, 1951, he was readmitted to hospital after an attack of acute retrosternal pain. An electrocardiogram showed evidence of recent myocardial infarction. In December, 1951, the liver and spleen were still enlarged and tests of liver function still gave abnormal results.

This was a case of infectious hepatitis with permanent impairment of liver function in an elderly man with coronary heart disease.

CASE 18.—Mrs. M.S., aged fifty-one years, a housewife, was admitted to hospital in August, 1951. She had been jaundiced for four weeks following pregnancies six and four years previously. After her second pregnancy she had never felt well, being easily tired and disinclined to eat. Shortly before her admission to hospital acute abdominal swelling developed. She was wasted and sallow, but not jaundiced. Numerous spider nævi were present on her face and neck and on the upper part of her arms. The abdomen was grossly distended with fluid. After tapping, the spleen was palpated, but the liver was not enlarged. On *paracentesis abdominis* five litres of straw-coloured fluid were withdrawn. She promptly lapsed into coma and was revived by transfusions of blood, serum and glucose (10%) and by an intragastric drip administration of five pints of milk daily. The ascites rapidly reaccumulated and she passed some melæna stools. After a second paracentesis, when the fluid was very slowly removed to prevent rapid reaccumulation, she lapsed again into profound coma and died.

At autopsy the liver was shrunken and the surface was studded with irregular nodules. Examination of sections revealed extensive fibrosis and a few islands of disorganized liver cells.

This was a case of chronic hepatitis, probably of post-infectious type, in a middle-aged woman. There is evidence in this case of the unfavourable influence of pregnancy on the clinical course of the disease.

Comment.—"Chronic hepatitis" is a loose term used to describe those cases in which the infection does not follow the usual self-limiting course (Wood, King *et alii*, 1948). In some instances (for example, Case 17) several relapses of jaundice may occur; but recovery with the restoration of normal liver function may take place. In other instances the characteristic picture of cirrhosis may develop (Sherlock, 1947), and in many of these cases a clear-cut history of initial infection may be lacking, suggesting an analogy with chronic nephritis. It may be of possible significance that three of the four patients in this group were females. One of the patients has died, and it is unlikely that the other two will survive for long. Remittent jaundice occurred in all cases, followed by a history of chronic ill health and manifestations of cirrhosis—the appearance of spider angiomata, splenomegaly, ascites or gastro-intestinal hæmorrhages.

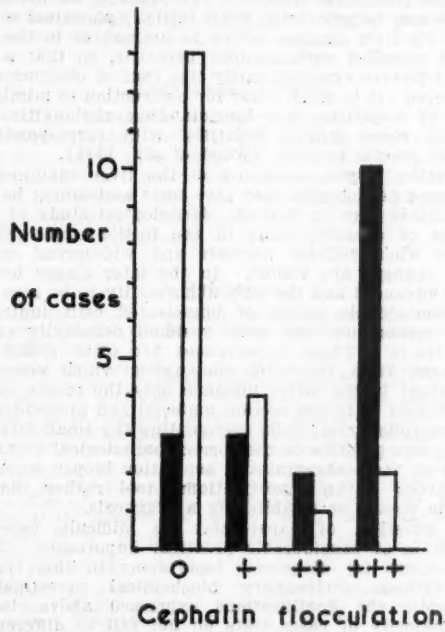

FIGURE VIIIA.

Results of cephalin flocculation test in 20 cases of infectious hepatitis and 20 cases of obstructive jaundice (due to stones or carcinoma of the pancreas). Black columns, infectious hepatitis; white columns, obstructive jaundice.

In our present state of knowledge it is impossible to differentiate post-infectious chronic hepatitis from nutritional cirrhosis on biochemical or histological grounds. The diagnostic findings in both diseases are strongly positive results to flocculation tests and reversal of the albumin-globulin ratio in conjunction with the histological appearances of extensive fibrosis surrounding islands of disorganized parenchymal cells.

It is possible to prolong the lives of some of these patients by means of intragastric and intravenous alimentation when liver failure threatens. In addition, Farquhar *et alii* (1950) have presented some experimental and clinical evidence that aureomycin exerts a protective action on the liver against hypothetical enterogenous toxins; this form of treatment is at present under investigation.

Biochemical Findings.

The cephalin flocculation test of Hangar is usually taken as an index of hepato-cellular damage, and is therefore of considerable importance in the diagnosis of jaundice. The findings in these 20 cases are shown in Figure VIIIA. As a "+" flocculation is of little pathological significance, in six cases the result of this test was not helpful in diagnosis.

The normal range of serum alkaline phosphatase is three to thirteen King-Armstrong units; in infectious hepatitis moderate elevation as a rule occurs (up to 30 units); and in obstructive jaundice levels of above 30 units are encountered. The values recorded in this group are shown in Figure VIIIB.

Results obtained with these two tests in these cases of infectious hepatitis and in a comparable group of patients with obstructive jaundice are compared in Figures VIIIA and VIIIB. It is apparent that, although the flocculation test result is positive in most cases of hepatitis and usually negative in obstructive jaundice, and that the figure of 30 units of phosphatase serves as an arbitrary dividing line in these types of jaundice, a certain amount of overlap occurs.

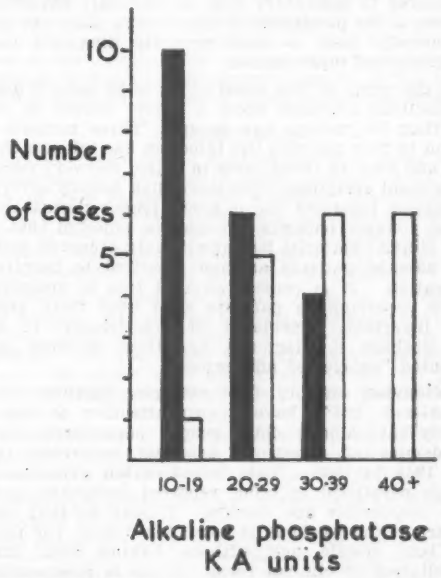

FIGURE VIIIB.

Level of serum alkaline phosphatase in 20 cases of infectious hepatitis compared with 20 cases of obstructive jaundice. Black columns, infectious hepatitis; white columns, obstructive jaundice.

Estimations of serum protein content are of value in the diagnosis of infectious hepatitis. In general a lower level of serum albumin is recorded in association with moderate elevation of the globulin content. This tendency is exaggerated in proportion to the severity or chronicity of the infection. Weiden (1952) has shown that quantitative estimations of serum γ globulin are helpful in the differential diagnosis of infectious and obstructive jaundice.

Radiological Studies.

Cholecystography is an investigation of little value in the presence of jaundice; but a flat X-ray picture of the gall-bladder is useful in excluding the presence of radio-opaque stones. Eleven patients in these series were radiologically examined shortly after their admission to hospital, with negative results. Ten patients returned to the out-patient clinic for cholecystography after their discharge from hospital, and in all cases normal excretion of the dye was observed.

Discussion.

Until the virus of infectious hepatitis is isolated and suitable serological tests can be employed, the diagnosis of infectious hepatitis will remain a matter of conjecture,

especially when, as so frequently occurs, no history of contact is obtained. For the purposes of this study this diagnosis has been accepted when the majority of the following features have been present in a case: exposure to the infection, gastro-intestinal prodromal symptoms, jaundice, typical biochemical test results, and histological evidence—either at biopsy or at autopsy—of the characteristic lesion of infection with the virus, and when, if any doubt remains, laparotomy discloses no cause of extra-hepatic obstruction.

This study indicates that infectious hepatitis is not a rare occurrence in elderly people and that it should always be considered in the differential diagnosis of jaundice in this age group. Clinically, infectious hepatitis at this age is not always so easy to recognize as in younger people, and recourse to laboratory aids is essential; nevertheless, awareness of the possibility of hepatitis in older age groups will assuredly lead to more accurate diagnosis and to fewer ill-advised laparotomies.

From the study of this small group of 20 cases it appears that infectious hepatitis takes a graver course in elderly people than in younger age groups. Three patients have died, and in four patients the infection has taken a chronic course, and even in those cases in which recovery occurred, constitutional symptoms were more than usually severe and the jaundice persisted for a much longer period than is usual in younger patients. It may be objected that selection of clinical material has unwittingly occurred and that mildly affected patients are not admitted to hospital for investigation. It is considered that this is unlikely, for jaundice occurring in patients aged over forty years is almost invariably considered by practitioners to be an urgent problem in diagnosis, and these patients in fact represented "unselected admissions".

Scandinavian authors (for example, Bjoenboe *et alii*, 1947; Alsted, 1947) have drawn attention to the high mortality rate among older people, particularly females, in epidemics of infectious hepatitis occurring in the period 1944 to 1946. This Scandinavian experience has not been paralleled in other reported outbreaks, and the factors responsible are obscure. It may be that only in that part of the world is it usual for women (or men) to reach late middle age without having been infected in subclinical or clinical form. If, as is presumably the case, the severe infections were in non-immune persons, we have to consider the possibility that, more than in other virus diseases, the outcome of infectious hepatitis is determined by age and by various nutritional or endocrine factors ruling at the time of infection.

There are interesting analogies which should not, however, be pressed too far between infectious hepatitis and poliomyelitis. In the very young, poliomyelitis is a relatively mild illness and the incidence of paralysis is low; but both the incidence of paralysis and the mortality rate rise in proportion to age. Infectious hepatitis is likewise thought to be a harmless illness in children (Capps *et alii*, 1952); but at the other extreme of life it would appear to carry a greater risk of death or chronicity.

The differential diagnosis of infectious hepatitis in this age group offers many practical difficulties; in the present series there was often little to be noted in the character of the pain which occurred prior to the onset of the jaundice to distinguish it from either biliary colic or the pain of carcinoma of the pancreas. Moreover, to make the matter more confusing, jaundice due to carcinoma of the common bile duct may occur early in the course of that disease and may be painless. Jaundice due to nutritional hepatitis is fairly common in this country, but does not appear to be a widely recognized syndrome. Joske and Turner (1952) have reviewed the clinical findings in 78 chronic alcoholic patients in Melbourne and found that jaundice was present in eight. In advanced alcoholic cirrhosis jaundice is common, but it has also been clearly demonstrated that severe fatty infiltration of the liver may also be associated with jaundice occurring in an alcoholic patient with a history of poor nutrition and with smooth enlargement of the liver.

Recourse to biochemical tests in diagnosis is therefore essential before surgery is contemplated, even if this means sending specimens of blood to a laboratory if these facilities are not available. Most reliance is placed on the cephalin flocculation test and on estimations of serum proteins and alkaline phosphatase. In infectious hepatitis the result of the cephalin flocculation test (or thymol turbidity test) is abnormal, some alteration in the pattern of serum proteins is detectable, while the alkaline phosphatase level is only moderately elevated. In obstructive jaundice from whatever cause, abnormality of the cephalin flocculation test result is uncommon, the pattern of serum proteins is not significantly altered, and the phosphatase level is nearly always above 30 units. However, no biochemical test is ever infallible, and the foregoing case reports show many deviations from this pattern. The jaundice of infectious hepatitis may be protracted while initially abnormal results to tests for liver damage return to normal, as in the rare cases of so-called cholangiolitic hepatitis, so that a biochemical picture extraordinarily like that of obstruction is encountered. It is much rarer for obstruction to mimic the picture of hepatitis; but long-standing cholangitis may result in more diffuse hepatitis, with correspondingly abnormal results to tests (Wood *et alii*, 1948).

Aspiration biopsy fragments of the liver examined by experienced pathologists may give some assistance; but the value of this may be limited. Histological study of liver tissue is of greatest value in the initial stages of the jaundice when cellular necrosis and widespread inflammatory changes are visible. In the later stages healing may be advanced and the only abnormality to be seen may be a questionable excess of binucleated cells indicating active regeneration and some residual cellularity of the portal tracts. These appearances are often difficult to distinguish from the mild cholangitis which occurs in obstruction; in the latter instance only the excess of bile pigment seen in frozen section material and preponderance of polymorphonuclear cells surrounding the small bile duct elements are pointers to the correct pathological diagnosis. In view of these uncertainties aspiration biopsy must still be regarded as an investigational tool rather than as infallible means of establishing a diagnosis.

The question of laparotomy in difficult cases of jaundice is of considerable practical importance. There is no excuse for diagnostic laparotomy in this type of case, without preliminary biochemical investigations, for despite the qualifications expressed above, in the great majority of cases tests do not fail to differentiate between infectious hepatitis and obstructive jaundice. Patients suffering from severe infectious hepatitis are "poor surgical risks", for the damaged organ is less able to detoxify anæsthetic substances and is most vulnerable to the anoxic effects of surgical shock. Surgical exploration is justifiable in doubtful cases when there is a negative response to the cephalin flocculation test and the alkaline phosphatase content is strikingly elevated. It is not overstressing the point to say that exploration should never be contemplated unless the results of the biochemical tests are available. These findings should then be reviewed by the physician and the surgeon in conjunction with the clinical features of the case.

Little is known of the epidemiology of infectious hepatitis in Australia, and there is a paucity of information on the severity of infection and the prognosis in different age groups; the information that does exist has been drawn from studies of patients admitted to hospital, which tends to give misleading impressions. In so far as the disease is of some economic importance by virtue of the extended duration of incapacity it causes, and as a significant proportion of cases end fatally or lead to chronic disablement, it is hoped that other States will follow the lead of Western Australia in making compulsory notification of infectious hepatitis to the public authorities.[1]

Summary.

Twenty patients—ten males and ten females—aged over forty years, suffering from infectious hepatitis, have been studied.

[1] Since this was written infectious hepatitis has been made notifiable in Victoria.

Manifestations of infection were more severe than in younger patients. In four cases (two deaths) the infection was severe and fulminant, and in four (one death) the infection caused several relapses and the development of advanced cirrhosis. Even in the average case the symptoms were severe and the jaundice was unusually prolonged.

Many patients had complained of severe right upper abdominal pain, which mimicked that of gall-stones or carcinoma of the pancreas.

Biochemical tests are necessary to differentiate infectious jaundice from that due to obstruction, and should be performed before surgery is contemplated. The cephalin flocculation and serum alkaline phosphatase tests supply the most valuable information.

Unless it is performed shortly after the onset of jaundice, aspiration biopsy of the liver is of limited value in the differential diagnosis.

Acknowledgements.

I wish to thank the honorary physicians of the Royal Melbourne Hospital for referring patients for investigation. I am also deeply indebted to my colleagues in the unit for undertaking the biochemical tests and for the interpretation of histological material. Mr. Matthaei, photographer of the University of Melbourne Faculty Workshops, kindly prepared the photographs.

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Legends to Illustrations.

FIGURE I.—Case 1: Liver biopsy taken one week after the onset of jaundice, showing diffuse necrosis and infiltration with inflammatory cells. (Hæmatoxylin and eosin stain, × 150.)

FIGURE II.—Case 8: Liver biopsy showing cellularity of the portal tracts and slight irregularity in size of liver cells. (Hæmatoxylin and eosin stain, × 600.)

FIGURE III.—Case 10: Liver biopsy showing deposition of bile pigment in the centrolobular zones. Arrows indicate "bile plugging". (Hæmatoxylin and eosin stain, × 150.)

FIGURE IV.—Case 11: Fulminating hepatitis. This section shows widespread necrosis of the liver cells. (Hæmatoxylin and eosin stain, × 150.)

FIGURE V.—Case 14: Liver biopsy taken four months after the onset of jaundice, showing excessive fibrosis and nodular hyperplasia. (Best's carmine stain, × 150.)

FIGURE VI.—Case 14: Liver biopsy taken one year after Figure V, showing total restoration on normal lobular architecture. (Best's carmine stain, × 150.)

FIGURE VII.—Case 17: Liver biopsy showing fibrosis and inflammation of the portal tracts and disorganization of the lobule. Slight fatty changes are seen in many liver cells. (Best's carmine stain, × 150.)

Reports of Cases.

NEUROGENIC TUMOUR OF THE STOMACH: CASE REPORT OF A PROBABLE NEURILEMMOMA.[1]

By THOMAS F. ROSE and EVA SHIPTON,
Sydney.

THIS is the history of a woman who developed a large neurogenic, probable neurilemmomal, tumour of the stomach, which was successfully removed by subtotal gastrectomy.

Clinical Record.

The patient was a female, aged seventy-eight years, who was first examined on November 14, 1949. She gave a six months' history of mild epigastric pain occurring immediately after meals. The pain would last for half an hour and then gradually subside; it was not relieved by alkaline powders or mixtures. There was no bleeding or vomiting, and she had lost no weight. Nothing relevant was found in the previous history.

Examination showed the patient to be a well-preserved elderly woman. The relevant physical finding was the presence of a firm, smooth, freely movable, rounded swelling in the epigastrium. It was large, measuring ten centimetres in diameter. It moved with respiration and did not pulsate. All other systems were normal, save for the presence of mild hypertension.

No manifestations of von Recklinghausen's neurofibromatosis were present. Examination of the urine revealed no abnormal constituents. A full blood count gave normal findings.

A barium meal examination was performed by Dr. Kristenson, who reported as follows:

> The stomach was normal in position and when filled with barium it showed very little abnormality but skiagrams taken with pressure applied to the abdomen showed that there were extensive filling defects along the greater curvature just proximal to the pylorus. The stomach, however, did not appear fixed and there was no particular tenderness over this area. The duodenal bulb was quite regular and showed no evidence of ulceration or abnormality. The stomach was completely empty five hours later.

The abdomen was explored on November 28, 1949, with the patient under general anæsthesia; a blood transfusion was given. A large, smooth, rounded tumour was found growing both exogastrically and endogastrically from the pyloric end of the greater curvature of the stomach. There were no surrounding adhesions or enlarged lymph nodes. As far as could be seen, the rest of the alimentary canal and abdominal organs were normal, no other tumours being present. As the tumour was so large, a partial gastrectomy was performed according to the method of Polyá with the jejunal loop antecolic and running from right to left.

Convalescence was uneventful, and the patient was very well when examined last in February, 1952.

Pathological examination of the specimen showed it to be a rounded, well encapsulated tumour, ten centimetres in diameter. Its cut surface had a grey, fibrous, whorled appearance which looked like a neurilemmoma or a leiomyoma. It appeared to arise from the submucosa of the stomach, and there was one small area of ulceration through the mucosa. It projected very little into the stomach lumen as compared with its exogastric mass. There were no other tumours in the stomach specimen. (Unfortunately, the specimen was destroyed inadvertently whilst awaiting photography, but the diagram in Figure I shows the relevant findings.)

Microscopic examination of sections showed the tumour to consist of a somewhat loose connective tissue stroma, through which were scattered numerous pleomorphic cells. Some of

[1] The spelling "neurilemmoma" is more correct than "neurilemoma" as originally coined by Stout (1935). The word is derived from two Greek stems, that for nerve (νεῦρον), and that for sheath (λέμμα).

these were round, some slightly ovoid, and others tended to be spindle-shaped, and of the last-mentioned some had long processes attached. No palisade formation was found. There was none of the fibrous tissue more characteristic of the neurofibroma. It was difficult to determine the exact nature of the growth, but it was probably of neurogenic origin (Figure II).

Discussion.

Canney (1948) has given an excellent discussion of these neurogenic tumours of the stomach. They arise from nerve sheaths, and there are two types, firstly, the neurilemmoma (Schwannoma), encapsulated and single, and secondly, the neurofibroma usually associated with other manifestations of von Recklinghausen's neurofibromatosis. This latter tumour is not so well encapsulated and has a greater tendency towards malignant change. Both Canney (1948) and Dick (1950) go into the histology of the stomach neurogenic tumours in great detail, so that it would be redundant to repeat it here.

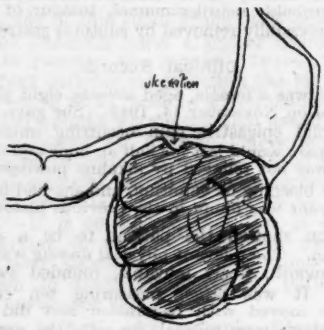

FIGURE I.

The neurilemmoma may be impossible to distinguish macroscopically from the leiomyoma, and even microscopically it may be very difficult unless special staining methods are used (Mallory, 1920; Golden and Stout, 1941; Willis, 1948), especially if the myoma has regimented nuclei (Willis, 1948).

Classification of this Tumour.

From the histological picture of this specimen (Figure II), it is obvious that it is a neurogenic tumour. It is a little more difficult to determine, however, whether it is a neurilemmoma or a neurofibroma. The neurilemmoma is well encapsulated, single and unassociated (except very rarely) with the stigmata of von Recklinghausen's neurofibromatosis. The neurofibroma is not so well encapsulated and tends to have much fibrous tissue throughout its substance (Canney, 1948; Dick, 1950). It may be one of several stomach or alimentary canal tumours and is almost always associated with generalized neurofibromatosis.

Consequently, we believe this tumour to be a neurilemmoma with Antoni Type B cells (Antoni, 1920).

Frequency of Neurilemmoma of the Stomach.

Apart from the small neurilemmomata in the stomach wall which are common incidental findings at autopsies (Willis, 1948*a*), neurogenic tumours of the stomach are rare (Canney, 1948), though not so rare as those of the duodenum (Rose, 1949). Few surgical text-books (including Aird, 1949) mention these stomach nerve tumours. Foot (1945*a*), Professor of Surgical Pathology at Cornell University, had seen none in the decade prior to the publication of his book.

Thompson and Oyster (1950) stated that in 20,000 autopsies there were 66 (0·33%) benign tumours of the stomach as opposed to 498 malignant tumours; that is, the benign tumours were 13·6% of the gastric neoplasms. In addition, these workers studied clinically 94 benign tumours, and of these only 28 (29%) produced symptoms. Eight of the benign tumours were called neurofibromata and another seven fibromata (which are usually of nervous origin when studied carefully). It is difficult to say from reading this analysis (as it is from reading many other articles on this subject) whether the term "neurofibroma" is used as being synonymous with "neurogenic tumour", no attempt being made to differentiate the neurilemmomata from the neurofibromata, or whether no neurilemmomata were found at all. Ranson and Kay (1940) stressed the fact that these are different tumours both in pathology and in behaviour. This is an important observation, because the neurofibroma is much more likely to become malignant than the neurilemmoma (Canney, 1948).

Age Incidence.

Owing to the confusion between these two tumours in the literature, it is difficult to ascertain a true age incidence of stomach neurilemmomata. Feyrter (1948) states that their frequency rises with age. Maggi, Meerof and Kuperman (1951) state that neurofibromata can occur from infancy to senescence. However, here again the term "neurofibroma" is not defined.

Diagnosis.

Clinically, there is no characteristic picture of stomach neurilemmomata (Gelin, 1950). There may be hæmorrhage, revealed or occult, if the tumour ulcerates, or obstructive symptoms due to a small pyloric tumour or to a large one in the body of the stomach. The patient may actually feel the tumour or merely have a history of epigastric pain, as in this patient herein reported. On the other hand, there may be no symptoms at all.

Physical examination usually reveals no relevant abnormality, or it may reveal a tumour which is freely movable. If the stigmata of generalized neurofibromatosis are present, a neurofibroma rather than a neurilemmoma is diagnosed.

As far as ancillary aids to diagnosis are concerned, a fractional test meal examination gives little help, as its results may vary from normal acid values to achlorhydria (Maggi, Meeroff and Kuperman, 1951). Gastroscopy may help to differentiate the neurilemmoma from a carcinoma, especially if the lesion is small, by showing the smooth mucosa over it often capped with its characteristic ulceration.

Radiologically, an opaque meal examination may reveal a filling defect and sometimes the crater niche due to the ulceration often present on the summit of the tumour. In this case report, it is interesting to note that although there was a large palpable tumour, the barium meal examination revealed little abnormality in the stomach until pressure was applied to the abdomen by the radiologist. Even then, the full extent of the tumour was not revealed, because the greater mass of the tumour was exogastric, and the small area of ulceration was not shown up by the examination.

With regard to the patient who is the object of this report, at her advanced age a tumour of the stomach is usually diagnosed as a carcinoma, as actually did occur. However, the surprising absence of loss of weight and the excellent general condition of the patient with such a large tumour were explained by the operative findings. Batey's case (1951) was also diagnosed as one of carcinoma. Gastroscopic examination might have helped furnish the correct diagnosis, but it was thought unnecessary to inflict this extra procedure on this elderly woman.

Treatment.

Though these neurilemmomata may be treated by local excision when small, one must remember that Tate and Fusaro (1948) showed that 10% of neurogenic tumours may become malignant. However, if they are large, as in this patient's case, one has no choice, as a partial gastrectomy is the easiest operation to perform. That this was justified here is shown when one learns that a follow-up twenty-seven months later revealed the patient at the age of eighty years to be healthy and symptomless.

Summary.

A case of a neurogenic tumour, probably a neurilemmoma, occurring in a female patient aged seventy-eight years is presented and discussed. This was treated by partial gastrectomy, the patient being alive and well two years and three months later.

Acknowledgements.

We wish to thank Dr. Frank Carmody, of Bondi Junction, for the clinical details of the patient prior to operation. We also wish to thank Mr. R. Johnson for the photomicrograph of the specimen.

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A CASE OF INTRACEREBRAL ARTERIO-VENOUS ANGIOMA CAUSING HEMIPLEGIA.[1]

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Miss M.C., aged twenty-three years, had suffered from poliomyelitis at the age of two years. This affected her left leg and foot, in which a tendon transplantation was subsequently carried out. She was perfectly well until the age of thirteen years, when her right leg and foot became weak and were dragged when she walked. A plaster of Paris cast was applied for six months, and as there was no improvement the foot was operated upon, without relief. About two years later the right arm and hand started to become weak and she developed a squint. The weakness had gradually progressed in the right arm and hand, and now there was practically no power in them at all. During the last two years she had had sudden attacks of blurred vision in the left eye.

On examination of the patient there was an external strabismus of the right eye. The right upper limb was almost completely paralysed and the fingers and thumb were held in a contracted position of flexion. There was partial spastic paralysis of the right lower limb, but the patient was able to walk. On auscultation of the head a systolic bruit could be heard over both mastoid regions. Plain skiagrams of her skull failed to reveal any abnormality, but arteriographic examination of the left internal carotid system revealed a huge arterio-venous aneurysm, arising from the middle cerebral artery and situated about the centre of the left cerebral hemisphere (see Figures I and II).

A pneumoencephalographic examination was subsequently carried out and showed that the arterio-venous malformation was situated deep in the cerebrum, but projected into the left lateral ventricle.

Although it was realized that a direct attack on this vascular abnormality would be fraught with considerable danger, it was thought that something should be done to check its growth and, if possible, prevent the occurrence of intracerebral and intraventricular hæmorrhage; thus it was decided to ligate the left internal carotid artery, in order to diminish the amount of blood entering it. In view of the extensive contractures already present, there did not seem to be much chance of any recovery of voluntary power in the right limbs.

Accordingly, at operation on April 16, 1951, the left internal carotid artery was exposed in the neck and clamped. An electroencephalographic examination was then carried out for thirty minutes, without the recording of any abnormal brain waves from the left cerebral hemisphere, and consequently this artery was ligated.

On the following day the patient developed a partial aphasia, of a nominal character only, in that she could not name familiar objects.

Treatment by intravenous injections of one-tenth of a milligramme of histamine every four hours and by injections of "Novocain" into the left stellate ganglion through a polythene tube in the neck was started and maintained for the next four days, during which time her condition improved, so that she was allowed to leave hospital on April 26, 1951. Her hemiplegia remained the same and the bruit was still present, though less loud.

Legends to Illustrations.

FIGURE I.—Lateral arteriogram, after percutaneous injection of "Uriodone" into the common carotid artery in the neck, showing the enormous collection of blood in the arterio-venous aneurysm in the middle of the brain.

FIGURE II.—Antero-posterior arteriogram showing the extent of the vascular abnormality in the left hemisphere and crossing the mid-line.

Addendum.

This patient was readmitted to hospital early in June, 1952. Her general condition had remained satisfactory, although the right hemiplegia had not changed. The systolic bruit could still be heard in her head, but it was not so loud as before. Arteriographic examination of the right internal carotid system showed satisfactory filling of the main arteries in both hemispheres and also showed that the arterio-venous aneurysm, though still filling from the right middle cerebral system, was smaller and less clearly defined as it contained less blood.

[1] This patient was shown at a clinical meeting of the New South Wales Branch of the British Medical Association, held on May 24, 1951, at the Royal Prince Alfred Hospital.

A FATAL CASE OF MURRAY VALLEY ENCEPHALITIS OCCURRING AT NARRABRI IN NEW SOUTH WALES.

By A. K. GARVEN and J. MARGOLIS,

The Royal Alexandra Hospital for Children, Sydney;

WITH A NOTE ON THE ISOLATION OF THE VIRUS

BY E. L. FRENCH,[1]

The Walter and Eliza Hall Institute and the Department of Experimental Medicine, University of Melbourne.

DURING the first three months of 1951 an outbreak of acute encephalitis was recognized in the Murray Valley district of Victoria (Anderson, 1952) and the causative organism isolated (French, 1952). The clinical features of the disease have been described by Robertson and McLorinan (1952) and extensive studies of its epidemiological features have been made (Anderson *et alii*, 1952). Serological evidence of widespread infection in human beings, horses and wild birds indicates that in the summer of 1950-1951 the virus was widespread in the Murray Valley area of northern Victoria. The likelihood that Australian X disease of 1917-1918 (Cleland, Campbell and Bradley, 1918) was due to the same virus makes the report of a case of Murray Valley encephalitis in one of the classical localities of Australian X disease of special interest.

The present paper reports a case of encephalomyelitis occurring at Narrabri in New South Wales about the middle of March, 1951, from which a virus identical with the virus of Murray Valley encephalitis was isolated.

[1] Working with the aid of a grant from the National Health and Medical Research Council, Canberra.

Clinical Record.

W.C., aged two years, was admitted to the Royal Alexandra Hospital for Children on April 16, 1951. For three days he had had a cold, but had not been unduly distressed until the morning of his admission to hospital, when he had a generalized convulsive seizure with both tonic and clonic movements. This was followed an hour later by a second convulsive seizure involving the right arm and leg, which was controlled with difficulty by sedation.

Physical examination of the patient on his admission to hospital revealed clonic movements of the right side of the face, the right arm and the right leg. The eyes deviated to the right and the pupils were dilated and did not react to light. There was no papilloedema. The abdominal reflexes were absent, the knee and ankle reflexes were exaggerated on the right side, and the right arm and leg were spastic. There was no neck stiffness and Kernig's sign was absent. The axillary temperature was 104° F. and remained between 104° and 106° F. throughout the course of the illness. Examination of the other systems gave normal results.

A lumbar puncture was performed and produced clear cerebro-spinal fluid with the following features: there were 30 leucocytes per cubic millimetre, 76% being polymorphonuclear cells and 24% lymphocytes; the total protein content was 20 milligrammes *per centum*; the chloride content was 730 milligrammes *per centum*; the glucose content was not decreased; the fluid was sterile. The pressure was not recorded.

The following day the child was still comatose. Slight neck and spine stiffness became apparent, the tendon reflexes were hyperactive and a Babinski response was elicited on each side. Repeated right-sided convulsions occurred, which ultimately failed to respond to "Avertin"; the child became cyanotic and died forty-eight hours after his admission to hospital.

The patient had lived at Narrabri all his life, about 200 yards from the Namoi Creek. The river had been in flood in November, 1950, and since then mosquitoes had been very troublesome. Three weeks before his illness his mother noticed that he had been severely bitten by mosquitoes, although other members of the family were apparently not affected.

A complete autopsy was performed. The body was that of a well-developed and well-nourished boy. On gross inspection the tissues of the central nervous system were normal. Apart from a slight degree of meningeal congestion, no macroscopic lesions could be detected in the brain or spinal cord externally and on the cut surfaces.

Histological examination revealed striking changes throughout the central nervous system, but these varied in intensity at different levels. Meningeal reaction was relatively slight and patchy, and where it was more obvious—as, for instance, in the occipital region—there was a mild degree of vascular congestion and some infiltration with polymorphonuclear cells, macrophages and lymphocytes, the last-mentioned cell predominating. Perivascular cuffing was also slight to moderate in degree, and occasional small perivascular hæmorrhages were present in the brain stem and the spinal cord.

The parenchymal lesions were widely distributed in the grey matter, although some areas of the brain escaped with relatively little obvious damage. The frontal lobes were the least affected, only occasional neurons showing changes of doubtful significance, such as intense staining of the cytoplasm and some nuclear pyknosis, whilst in some nerve cells the nuclei appeared to be swollen and faintly stained. The only definite lesions in this area were several small, widely separated islets of interstitial necrosis infiltrated with phagocytic cells. Otherwise the cellular reaction was minimal and consisted of a slight general increase in microglia.

In contrast to those mentioned above, the changes were most striking in the occipital cortex, where destruction of the majority of neurons was accompanied by intense inflammatory infiltration. In considerable areas of tissue practically every nerve cell was affected by an eosinophilic type of necrosis, the cytoplasm and often the nucleus having acquired a bright pink staining reaction. Other ganglion cells were shrunken, with irregular pyknotic nuclei, whilst still others were obviously in the last stages of disintegration; here and there neuronophagocytosis could be observed.

In the mid-brain and medulla vascular congestion was pronounced and small perivenous hæmorrhages were sometimes present. The perivascular cuffing was also heavier than in the cerebrum. The grey matter of the brain stem nuclei was extensively affected, but the changes tended to have a more patchy distribution. In these areas numerous polymorphonuclear and microglial cells were present, and the neurons suffered varying degrees of damage. Many small foci of interstitial necrosis were so closely packed with phagocytes that they resembled miliary abscesses.

Although the changes in the cerebellum were not quite so dramatic, the cortex was obviously involved. A considerable proportion of Purkinje cells showed various degrees of chromatolysis, pyknosis, cytoplasmic vacuolation and lysis. Here also occasional examples of neuronophagia were encountered, and in some folia small groups of Purkinje cells had more or less completely disappeared. In those areas accumulations of phagocytes were often quite dense, and these cells were spreading outwards into the molecular layer (Figure I) and sometimes indenting the granular cell layer.

The most conspicuous lesions were seen in the spinal cord, where hardly any normal neurons remained in either the anterior or the posterior horns of the grey matter, the former being rather more extensively involved. The cellular reaction was most striking, dense clusters of phagocytes marking the outlines of disappearing nerve cells, and where the necrotic neurons still persisted there was often a dense ring of polymorphonuclear cells and microglia invading their bodies from the periphery (Figures II and III).

Discussion.

In the light of the published reports of pathological appearances in the Murray Valley encephalitis epidemic (Robertson, 1952), some aspects of this case deserve particular attention.

Death having occurred only two days after the onset of encephalitic symptoms, the material examined shows earlier and more acute lesions than the specimens from other patients, who had survived for at least nine days and in most cases much longer. Hence, individual variation being granted, certain differences between the lesions described here and those reported in other cases are consistent with the various stages of disease at which the organs were examined.

On the whole, the distribution and the general pattern of damage were similar to other cases of Murray Valley encephalitis and also resembled those of Australian X disease (Cleland *et alii*, 1919), and Japanese B encephalitis (Zimmerman, 1946). The cerebellar lesions were not so advanced as in the examples described by Robertson (1952). In a high proportion of his cases Purkinje cells had disappeared over a wide area of cortex, whereas in the present case these cells could still be seen in various stages of degeneration, and relatively few had totally vanished, which is in keeping with the more acute course of the disease.

On the other hand, the changes found in the spinal cord closely resembled those of early poliomyelitis. The phenomenon of neuronophagia deserves special attention, as this was not described in other cases of the 1951 outbreak, although it appears to be present in the photomicrograph of the spinal cord section (published by Robertson, 1951). Indirect evidence suggests that this is probably a process which takes place in the first few days of illness and is rapidly completed. It is uncommon in cases of poliomyelitis in which death occurs more than three days after the onset of symptoms (Bodian, 1949), and whilst this does not necessarily apply to other virus conditions, the fact that neuronophagia is described in Australian X

TABLE I.

Results of Inoculating Mice of Different Ages in Groups of Five with Various Portions of Brain.

Age of Mice at Time of Inoculation.	Inoculum.				
	Cerebral Cortex.	Basal Ganglion.	Medulla.	Pons.	Cerebellum.
21 days	S S S S S[1]	8 8 9 9 10	S S S S S	10 11 S S S	11 11 12 S S
2 to 4 days ..	9 10 S S S	6[1] 6 6 7 8	S S S S S	9 9 10 S S	8 8 9 S S

[1] "S", survived for twenty-eight days after inoculation; "6", or other numbers, number of days of survival after inoculation.

disease, which is considered to be closely related if not identical with Murray Valley encephalitis, suggests that such a comparison is not unreasonable. In further support of this conclusion is the fact that in the 1917-1918 epidemic the average duration of fatal illness was only four to six days, only one of the 94 patients who died surviving for as long as twelve days and only a small number for nine days. This contrasts sharply with the present epidemic, in which the disease had a much longer course. It may also be noted that neuronophagia was much more obvious in the spinal cord and in particular in the lumbar enlargement, where the bodies of motor neurons could, perhaps, be expected to persist longer by virtue of their large size, whereas the remains of the smaller cells of the cerebral and cerebellar cortex seemed to be more rapidly removed.

It appears, therefore, that complete examination of the brain and spinal cord, and of the cerebellar cortex in particular, may be necessary to arrive at a histological diagnosis of Murray Valley encephalitis, in which the distribution rather than the finer details of the lesions may furnish the clue in distinguishing this condition from acute poliomyelitis.

Isolation of the Virus.

(E. L. French.)

Portions of brain were sent to Melbourne from Sydney by air, initially packed in dry ice; but on their arrival the refrigerant was spent and the specimens had thawed out. The various pieces of brain were emulsified separately by being ground with sterile alundum and made into a 20% emulsion with sterile nutrient broth (pH 7·4). This mixture was centrifuged for ten minutes at 3000 revolutions per minute in a laboratory angle centrifuge. To each one millilitre of the supernatant 4000 units of streptomycin and 750 units of penicillin were added and the mixture was inoculated intracerebrally into twenty-one day old mice and into suckling mice two to four days old. In addition to mice, developing chick embryos were inoculated on the chorio-allantoic membrane by the method of Beveridge and Burnet (1946).

The mice were inspected daily and the brains removed aseptically from the mice which died with signs of encephalitis. The typical symptoms of Murray Valley encephalitis infection in mice have been described previously (French, 1952). Table I shows the results of these inoculations.

By passaging of the brains of dead mice in further mice, fatal encephalitis developed between the sixth and ninth days in all the inoculated mice.

The eggs inoculated on the chorio-allantoic membrane were opened after three days' incubation at 35° C., and all membranes inoculated with each section of the brain were found to have typical pocks (between five and fifteen on each membrane). Serial passage of these membranes in eggs produced a copious growth of virus, which killed the embryos between forty and sixty hours after inoculation.

Relationship of the Virus Isolated to the Virus of Murray Valley Encephalitis.

The behaviour of the virus in chick embryos and in mice was similar to that found with the Victorian strains of Murray Valley encephalitis virus (French, 1952).

The virus was examined serologically by means of the complement fixation test. Complement-fixing antigen was prepared from infected chorio-allantoic membranes as described by French (1952). This material and a similar reagent prepared from the Victorian strain of Murray Valley encephalitis virus was set up in a complement fixation test with sera prepared by inoculating guinea-pigs intracerebrally or twenty-one day old mice intraperitoneally with the virus under examination. The serum was collected twenty-one days after inoculation of the animals. The complement fixation technique used was an overnight cold fixation technique slightly modified from that used by Anderson *et alii* (1952). The results are set out in Table II.

TABLE II.

Complement Fixation Tests with the Victorian and New South Wales Strains of Murray Valley Encephalitis.[1]

Serum Dilutions.	Victorian Strain Antigen.				New South Wales Strain Antigen.			
	1:2	1:4	1:8	1:16	1:2	1:4	1:8	1:16
P376[2]:								
1: 10	++++	++++	++++	++++	++++	++++	++++	++++
1: 20	++++	++++	++++	+++	++++	++++	++++	+++
1: 40	++++	++++	++++	++	+++	++++	++++	+
1: 80	++	++++	+++	−	++	++++	++	−
1:160	−	++	+	−	−	+	−	−
1:320	−	−	−	−	−	−	−	−
M4492[3]:								
1: 10	++++	++++	++++	+++	++++	++++	++++	+++
1: 20	++++	++++	++++	++	++++	++++	++++	+++
1: 40	++++	++++	+++	−	++++	++++	++++	++
1: 80	++++	++++	++	−	++++	++++	+++	+
1:160	+	±	±	−	+	±	±	−
1:320	−	−	−	−	−	−	−	−

[1] Serum controls consisted of dilutions of both types of serum put up as shown in the table, with dilution of 1:2 to 1:16 of normal chorio-allantoic membrane suspension. These gave negative results throughout. "++++", complete fixation of three hæmolytic doses; "−", no fixation; "++", 50% hæmolysis, taken as the end-point.

[2] Guinea-pig serum prepared against the Victorian strain of Murray Valley encephalitis virus.

[3] Pooled mouse serum prepared against the New South Wales strain of virus.

Discussion.

The isolation of the virus from this case provides a number of points for discussion. It is of interest that the virus withstood a period without refrigeration during its transmission by post from Sydney to Melbourne. This would seem to indicate that this virus is fairly stable when thus transported in brain tissue, or that a particularly high concentration of virus was present in this case.

In the two Victorian cases of Murray Valley encephalitis from which a virus was isolated, a good growth of virus was obtained on primary inoculation of the brain onto the chorio-allantoic membrane of the developing chick embryo, but no virus was isolated by inoculation of mice (French, 1952). In the present case, as can be seen from Table I, the virus was also readily isolated by inoculating mice intracerebrally. This result in mice was obtained probably because the child died before much antibody capable of neutralizing the virus in mice had been produced. The possible part played by such antibody in the isolation of virus in mice and in eggs has been discussed previously (French, 1952). However, the advantages of the chorio-allantoic membrane of developing chick embryos over mouse inoculations as a means of isolating Murray Valley encephalitis virus is further demonstrated in this case. Both suckling mice and twenty-one day old mice inoculated with the medulla from this subject failed to develop encephalitis, whilst the eggs inoculated with a portion of the same suspension readily yielded virus. Furthermore, twenty-one day old mice inoculated intracerebrally with cerebral cortex failed to show any reaction, and only two out of five suckling mice died with encephalitis, whilst eggs inoculated with a portion of the same suspension were found to have an average of eight specific pocks per membrane.

The results of the complement fixation tests with the Victorian and New South Wales strains of virus set out in Table II reveal the complete homogeneity of the two strains as far as the complement fixation test is concerned. When the result is considered along with the reactions in mice and in chick embryos, this is thought to be sufficient evidence for deciding that the virus isolated is a strain of Murray Valley encephalitis virus.

The epidemiological studies of Anderson *et alii* (1952) and of Anderson and Mackerras (1952) have established the widespread distribution of the Murray Valley encephalitis virus throughout the eastern portion of Australia during or prior to the summer of 1950-1951. These workers found complement-fixing antibody to Murray Valley encephalitis virus in the sera of humans and horses along the eastern portion of Australia from Cairns in the north to the Victorian Great Dividing Range in the south. The coastal belt of New South Wales appeared to be free of the virus, but inland along the courses of the Lachlan and Darling Rivers antibody was found to be present in both human and horse sera.

Narrabri is situated on the Namoi River, which is a tributary of the Darling River and was one of the centres of Australian X disease (Cleland *et alii*, 1918). It is about 400 miles north of the River Murray. The serological findings of Anderson and his colleagues, and the isolation of the Murray Valley encephalitis virus from an acute case in this area suggest that this virus was present in the area during the summer of 1950-1951. It is not unlikely that the outbreak in this area in 1917 of Australian X disease, which resembles Murray Valley encephalitis both clinically and in its histopathology in the human brain (Robertson and McLorinan, 1952; Robertson, 1952), was caused by a virus very similar to, if not identical with, the Murray Valley encephalitis virus.

Summary.

1. A case of encephalitis in a child, aged two years, living at Narrabri, New South Wales, is described. The child was mildly ill with a "cold" for three days before his admission to hospital with generalized convulsions. He died forty-eight hours after his admission.

2. Histological examination revealed diffuse encephalomyelitis and patchy meningitis. The lesions are briefly described and illustrated.

3. A virus serologically identical by the complement fixation test with the virus of Murray Valley encephalitis was isolated in developing chick embryos and in mice from the brain obtained *post mortem*.

4. The significance of this case of Murray Valley encephalitis occurring 400 miles from the River Murray is briefly discussed.

Acknowledgements.

We wish to thank Dr. S. E. L. Stening for permission to publish this article, and Dr. R. D. K. Reye for his advice and criticism in the preparation of the clinical and pathology report. The interest of Sir Macfarlane Burnet, F.R.S., in this work, and his criticism, are gratefully acknowledged.

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Legends to Illustrations.

FIGURE I.—Cerebellum (hæmatoxylin and eosin stain, × 100). Necrotic Purkinje cells with accompanying cellular reaction.

FIGURE II.—Lumbar part of the cord (hæmatoxylin and eosin stain, × 100). Extensive neuronal necrosis with intense inflammatory reaction and moderate perivascular cuffing.

FIGURE III.—Neuronophagia; ganglion cell seen in the centre of Figure II. × 450.

SPOROTRICHOSIS.

By B. B. BARRACK,

Honorary Dermatologist, Mater Misericordiæ Hospital, Brisbane,

WITH A REPORT ON THE MYCOLOGY BY

R. E. POWELL,

Queensland Institute of Medical Research, Brisbane.

SPOROTRICHOSIS in Australia is a rare disease. Robinson and Orban reported a case in October, 1951; prior to that no cases had been reported, although some may have been recognized. Molesworth stated in 1944 that it had never been reported in Australia.

The following case is of interest if only because of its rarity.

Sporotrichosis is a chronic mycotic infection which produces granulomatous swellings of the skin and subcutaneous tissues; exceptionally it may become systemic.

It is caused by a *Sporotrichum Schenckii* (Matruchot, 1910), of which there are several varieties. The organisms exist as a saprophyte on plants and grasses and possibly on certain small animals and insects. In tissue it resembles *Histoplasma capsulatum* in that the cells are yeast-like.

A number of cases have been reported in South Africa in miners, and in the United States of America in gardeners and florists. It also occurs in France in agricultural workers in a disseminated subcutaneous form.

Several clinical types are described, the most common being the localized lymphangitic type. Less common are the disseminated cutaneous and the systemic types.

In the localized lymphangitic type, to which this case belongs, the organisms gain entry through a small puncture or abrasion, usually on a finger or thumb. In about a week or more a primary lesion or sporotrichotic chancre develops. This may be in the form of a nodule or a superficial ulcer—there is generally more or less induration. After a latent period of a few days to several weeks or more nodules develop along the course of the lymphatics. The linear distribution of the nodules is characteristic of the infection. They are usually painless, and the adjacent lymph glands are not involved. The nodules assume a rosaceous tint and tend to break down, discharging viscoid greyish-yellow pus. These lesions tend to persist indefinitely, although cases have been reported in which they have healed spontaneously. There are usually no subjective symptoms, and in the literature this lymphangitic type appears to remain a localized process, rarely becoming systemic.

Differential diagnosis would involve other mycotic infections, syphilis, tuberculosis and pyogenic infection. The linear distribution, absence of pain and non-involvement of the glands would suggest the possibility of sporotrichosis.

In August, 1951, Dr. K. G. Lawrence, of Lismore, referred the patient to me with the following history. He had first interviewed the patient on July 4, 1951, when she had a history of a sore on the middle finger of her right hand for weeks. Examination of the patient had revealed an infected sore on the dorsum of her middle finger and multiple indurated areas about the size of a pea or smaller occurring in a line up the dorsum of her forearm. A provisional diagnosis of a low-grade infection had been made and a course of sulphadiazine and "Distaquane" given with no apparent benefit. On July 16 a nodule had been excised from the forearm and sent to a pathologist, who reported the presence of "diffuse chronic inflammation". At that stage there was practically no obvious inflammation in any of the nodules. On July 23 the area from which the nodule had been excised was well healed, but at the same time the nodule on the dorsum of her hand had broken down and was discharging pus. On August 16 practically all the nodules had broken down and had the appearance of granulomata. The patient was then sent to the laboratory for the taking of smears, culture of material *et cetera*, and the following report was given:

> (1) Cultures were attempted from the eroded areas and by puncturing the unbroken lesions on various media (blood agar, Sabouraud media and plain agar). No growth at 48 hours. At 4 days a total of about seven colonies only developed on blood agar and Sabourauds. These colonies were large and whitish in colour—strained filters of such showed the presence of a yeast or yeast-like cell, but no other organism.
>
> (2) Slides made direct from the lesion showed no yeast cells and no bacteria.

Dr. Lawrence stated that in the opinion of the laboratory it was impossible to determine whether the organism was contaminant or not.

I saw the patient on August 28, 1951, when she had the lesion described by Dr. Lawrence. She stated that she remembered pricking her finger on which the original lesion developed. The picture was so typical of sporotrichosis that a provisional diagnosis of sporotrichosis was made, and I referred her to the Queensland Institute of Medical Research for culture.

The patient returned to Lismore on the same day, after prescription of a potassium iodide mixture, the dosage being 10 grains three times a day, increasing daily up to 30 grains or more. Local treatment prescribed consisted of compresses of perchloride of mercury solution (1:5000) followed by glycerine with ichthyol in 10% solution.

Dr. Lawrence reported to me on October 8, 1951, that all lesions had healed.

No photograph was obtained of the original lesion owing to the short visit of the patient to Brisbane; however, a photograph of the scars has been obtained to show the distribution (Figure I). It was not possible to have a blood test carried out at the time, but later a Kahn test was carried out and the result was negative.

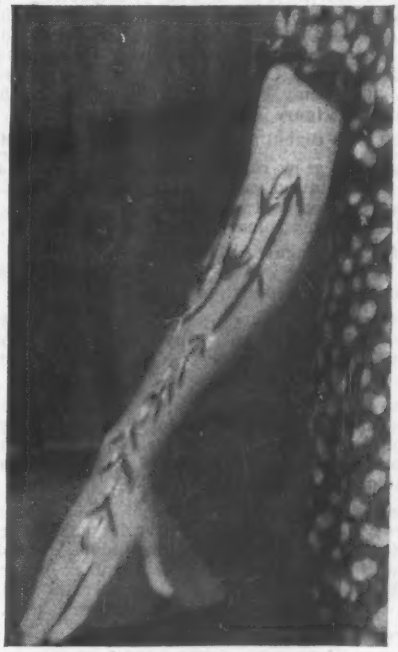

FIGURE I.

Arm of patient showing scars; arrows indicate the sequence of infection.

I am indebted to Dr. Lawrence for the excellent history and for his help and cooperation in gathering details of the case for me.

In regard to the reports of the pathologist and laboratory mentioned in Dr. Lawrence's letter, I should like to explain that the pathologist's report was perfectly correct, as he was reporting on one of the nodules excised and preserved in formalin. The laboratory probably grew the organism, but without clinical direction to search for this particular organism they were unlikely to identify it.

Mycology.

(R.E.P.)

At the Queensland Institute of Medical Research an unbroken nodule was punctured aseptically and the pus was inoculated directly onto Sabouraud's glucose agar slopes; then smears for microscopic examination were made. The Sabouraud's slopes were incubated at 30° C. and after five days numerous small, dull brown, yeast-like colonies were found to be growing on the surface of the agar. On growing, these quickly produced pale myceloid margins, one millimetre wide, while the centres became glistening, wrinkled and dark brown.

Examination of smears of the pus stained with hæmatoxylin and eosin, Giemsa and Gram-Weigert stains showed no evidence of pathogenic fungi.

Cultures of the organism were maintained on Sabouraud's glucose agar at 30° C., while the morphology was studied from slide cultures on corn meal, malt and Czapek's agar. Though the amount of growth varied appreciably on these three agars, there was little variation in the morphology.

The original yeast-like colonies were found to consist mainly of elliptical, budding, yeast-like cells measuring 2·0μ to 3·0μ by 3·0μ to 4·0μ, with a few fragments of mycelium (Figure II).

In the mycelial form the aerial hyphæ are scant and prostrate, measuring 1·5μ to 2·0μ in diameter. They are septate and well branched, bearing conidia apically or laterally on short lateral branches or on the main hyphal strands. The conidia are oval to pear shaped, measuring 2·0μ to 3·0μ by 3·0μ to 5·0μ, and are mainly borne on denticles. They may be borne singly on the hyphæ or in dense clumps (Figure III).

No traces of a sexual stage could be found in culture.

The organism grew rapidly on most media and retained its dark pigmentation on all synthetic and plant extract media, but on beef extract and blood agar the colonies were a dirty creamy colour.

On beef extract agar, growth was very flat and scanty; that on blood agar was far more extensive, the colony being wrinkled and glistening.

On potato-dextrose and malt agar the growth was luxurious, with abundant greyish-brown woolly aerial mycelium. The margins were pale and the reverse dark brown in all cases.

Corn meal, however, gave a somewhat sparse growth resembling more the colony type on Sabouraud's agar. The growth on Czapek's agar was dark, but flat and sparse. Growth on nutrient gelatine was slow, the gelatine being slightly liquefied after three weeks.

After maintenance of the organism on Sabouraud's agar for several months, the colony lost its glistening appearance and now grows with a fine, fluffy, greyish-brown, aerial overgrowth. The reverse is still dark brown in both young and old colonies, but in the latter the agar is discoloured, tending to become a dirty brown colour.

The organism was found to grow over a wide range of temperatures, the optimum being between 20° and 30° C. It grew poorly at 10° and 37° C., and was killed within seven days when incubated at 45° C.

The fungus was classified as *Sporotrichum Schenckii* var. *Beurmanni* (Matruchot and Ramond, 1905; Dodge, 1935).

Synonyms are *Sporotrichum Beurmanni* (Matruchot and Ramond, 1905; Dodge, 1935), *Rhinocladium Beurmanni* (Vuillemin, 1911), *Sporotrichopsis Beurmanni* (Gueguen, 1911) and *Rhinotrichum Beurmanni* (Ota, 1928).

Pathogenicity tests for the *Sporotrichum* strain isolated were carried out on white rats and on the chorio-allantoic membrane of the chick.

Rats were inoculated intraperitoneally with one-millilitre quantities of a heavy spore suspension. Rats were killed from eleven to twenty-eight days after inoculation, and the whole of the visceral cavity was examined, cultures, smears and sections being made of all the organs.

At the end of eleven days there were no apparent lesions or nodules in the viscera, but the organism was grown from the mesentery. This, however, was believed to be the residue of the original inoculum, as it was not isolated at later dates from the mesentery. At the end of twenty-five days nodules, one to two millimetres in diameter, were found studded in the membrane surrounding the testicles, and the organism was recovered in culture from this source. No nodules were found in the remainder of the peritoneum, but in one rat lesions were found on the large intestine. After surface sterilization of the infected intestinal area (with mercuric chloride) the organism was isolated in culture.

However, the pathogenic phase of the organism could not be found in smears or sections of any of the infected regions.

On the chorio-allantoic membrane of the chick a few pocks were produced on twelve-day eggs when inoculated with spore suspensions of the *Sporotrichum* strain. Cultures of the organism were grown from pocked membranes. In section the lesions consisted mainly of proliferation of the membrane tissue, but the parasitic *Sporotrichum* cells were found packed in macrophage cells. They were identical with forms described by Kligman and Baldridge (1951), being more or less globose, encapsulated, yeast-like forms 2·0μ to 7·0μ in diameter (Figure IV).

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Legends to Illustrations.

FIGURE II.—Yeast-like cells of *Sporotrichum Schenckii*, showing budding. × 2200. (Gram stain.)

FIGURE III.—Slide culture of *Sporotrichum Schenckii*, to show conidial formation. × 525. (Unstained.)

FIGURE IV.—Parasitized cells in the chorio-allantoic membrane of the chick. × 2200. (Hæmatoxylin and eosin strain.)

BILATERAL CYSTIC OVARIES (STEIN'S SYNDROME).

By RICHARD FLYNN,
Sydney.

IN an annotation in the *British Medical Journal* of November 10, 1951, a discussion of Stein's syndrome appears and references to the relevant literature are given. In one of these the essential features of the syndrome are stated as follows:

> The characteristics of this syndrome are menstrual irregularity featuring amenorrhœa, a history of sterility, masculine type of hirsutism, and, less consistently, retarded breast development and obesity. The ovaries are similarly and simultaneously enlarged and show typical cystic changes which are irreversible and refractory to hormonal therapy. The development of this condition is not congenital; neither is it inflammatory nor degenerative. It is the result of a definite endocrine disturbance.

At the time of Stein's last report in 1949, 76 patients examined in private practice over a period of twenty years had been operated on for this condition. The chief complaints were amenorrhœa and sterility. Prior to surgical treatment 48 patients were married and 28 single. Menstrual function was satisfactorily restored in all the single patients and in 90% of the married group. Forty-one patients in the latter group complained primarily of sterility. Twenty-seven, or 66%, conceived one or more times after surgical treatment.

Leventhal and Cohen (1951) summarize their paper as follows: (i) There is a definite clinical syndrome associated with bilateral polycystic ovaries, and characterized by menstrual irregularity, featuring amenorrhœa and sterility. Hirsutism is a frequent associated finding. (ii) The changes in the ovaries are refractory to all present forms of hormonal therapy. (iii) Restoration of normal menstrual and reproductive function follows bilateral wedge resection of the polycystic ovaries. (iv) Considerable hyperplasia of the *theca interna* layer is a constant finding in sections of the ovarian tissue removed. Luteinization of this layer is a frequent finding (Figure V). (v) A working hypothesis for the pathogenesis of this syndrome is presented.

Clinical Records.

Two cases of the syndrome are here reported.

CASE I.—J.C., a female patient, aged twenty-five years, was referred to me by Professor B. T. Mayes because of the growth of hair on her face and abdomen during the previous five years. She appeared older than her stated age, there was a heavy growth of hair on her face, and her body was covered with an acne-like rash. She also had gained much weight during the preceding five years. Her menstrual periods were regular, the last one having occurred five days prior to consultation. She had two living children, one two years of age and the other nine weeks old. She had had one miscarriage.

Physical examination of the patient revealed abnormal distribution of hair. Her blood pressure was 130 millimetres of mercury, systolic, and 80 millimetres, diastolic. X-ray examination of her skull and chest and excretion urography failed to reveal any abnormality. The basal metabolic rate was —20%. The ketosteroid excretion was estimated by Dr. Emily Day as 7·1 milligrammes per twenty-four hours.

On account of the pronounced hirsutism it was decided to explore her abdomen through a low mid-line incision. Both ovaries were enlarged and cystic, and there were numerous translucent cysts on the surface of both ovaries. The areas intervening between the cysts were a peculiar mottled colour—green areas on a pink background. (The ovaries in this patient were really an exact duplicate of the next patient's ovaries.) The adrenals felt normal. Because of her recent pregnancy it was thought that the ovaries were functioning normally, and to remove them might upset her endocrine balance.

In answer to my letter of inquiry on February 18, 1952, she replied as follows:

> In reference to your letter I received I am replying to same. My general health is about the same as before. My periods are normal every 28 days for about 6 days. I have no pain. A miscarriage in Oct. 1948 about 6 weeks pregnant. I gave birth to a baby daughter 5 lb. 3¼ ozs. at Royal Paddington. I thank you for your interest.

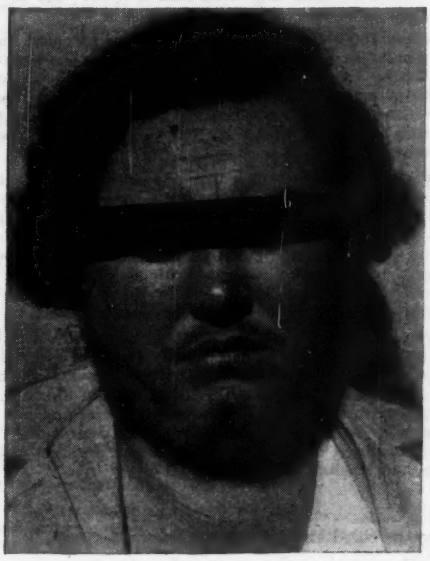

FIGURE I.

CASE II.—D.J., a female patient, had been a normal woman till about nine years before. She was fat, and had been so all her life. However, she had no facial growth of hair, and her menstrual periods came regularly once a month. She married and had a child. Although she has tried she has been unable to have another. Some time after the baby was born she noticed increasing obesity; she never worried about this, however, as all her family are fat. Then her menstrual periods became irregular, and at one stage she believed herself pregnant. This irregularity has progressed, and now the periods are spaced at intervals of months and last only about a day. There is a scanty reddish discharge. She experiences no pain with them. About four years ago she began to notice excessive growth of hair on her face and the lower part of her abdomen. She does not pass excessive amounts of urine, nor does she suffer from *pruritus vulvæ*. She has no headaches and does not become excessively breathless, and her eyesight and hearing are good.

Physical examination of the patient revealed an excessive growth of fairly stiff dark hair on the "beard region" (Figure I). The escutcheon was of male type. The forearms bore an excessive growth of stiff hair, but the legs were not remarkably hirsute. Her blood pressure was 235 millimetres of mercury, systolic, and 135 millimetres, diastolic. X-ray examination of the pituitary fossa revealed no abnormality. An excretion urogram was reported on as follows:

> The plain ray shows the kidneys to be quite normal in size and position for a patient of this age. The intravenous urogram shows there to be a double pelvis and double ureter on the right side but no definite evidence of suprarenal tumour can be seen.

A sugar tolerance test gave the following figures: before ingestion of glucose, 79 milligrammes *per centum*; half an hour after the ingestion of glucose, 141 milligrammes *per centum*; one hour after, 150 milligrammes *per centum*; one and a half hours after, 124 milligrammes *per centum*; two hours after, 97 milligrammes *per centum*. An electrocardiogram revealed no gross changes, but a faint slurring of the *R* waves in leads II and III and a flattened *CF* in lead IV were indicated. The 17-ketosteroid excretion was 5·46 milligrammes per-twenty-four hours. A concentration of a twenty-four-hour specimen of urine was injected into a rabbit, which died twenty-four hours later. No hæmorrhagic follicles or *corpora lutea* were present in either ovary. A urea concentration test showed that the kidneys were functioning normally. A general anæsthetic was given and a vaginal examination carried out; no abnormality was detected. Then a lower mid-line incision was made and a Maguire's retractor inserted. The uterus and adnexæ were exposed and a most unusual sight was beheld. The uterus was

FIGURE II.

normal, as were the Fallopian tubes. Both ovaries were elongated and whitish, somewhat soft in consistency, with numerous small dark cysts beneath the capsule. They were about two to two and a half inches long and resembled testes. The right ovary was removed by ligating the meso-ovarium with aneurysm needles and cutting. The appendix was then removed. The adrenals were apparently normal on palpation.

Dr. G. Davies's report on the ovary removed was as follows: The specimen consisted of a large ovary, which weighed 29 grammes and measured 7·5 by 2·7 by 2·0 centimetres. The external surface was smooth, white and very slightly lumpy (Figure II). Many blue spots corresponded to cysts, which approached close to the surface. At one end were four small cysts, each two or three millimetres in diameter, which projected above the level of the surrounding tissue. When a section was cut through the specimen, the cut surface was seen to be mainly white and glistening. There were many small cysts just beneath the surface, and each of these was filled with

transparent material, now coagulated by formalin. Minute congested blood vessels radiated from the hilum (Figure III). On microscopic examination of the ovary, the stroma was more abundant and more fibrous than usual. Sections included a number of Graafian follicles in various stages of development, and a number of these were atretic. In nine large follicles (Figures IV and V) there was degeneration of the granulosa layer, suggesting that the follicles were atretic; yet in three of them a cumulus, with or without an ovum, still remained. In some areas the stroma was less fibrous and much more cellular. The cells resembled the usual ovarian stroma cells, but were larger and less regular in size. In one section there were cells which resembled lipoid histiocytes (Figure VI).

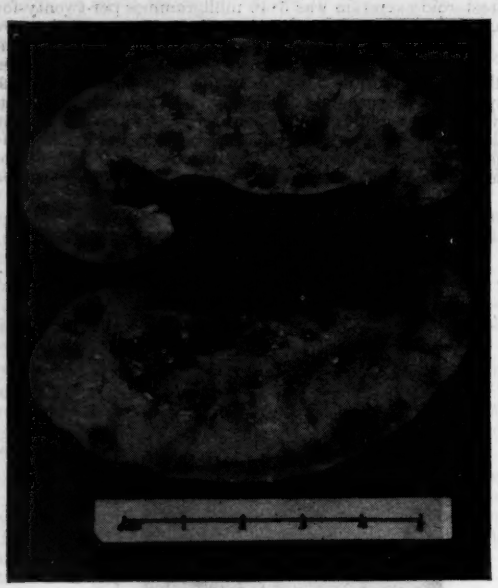

FIGURE III.

They were spherical and had abundant granular and foamy cytoplasm. There was no trace of *corpus luteum* either on macroscopic or on microscopic examination, and no definite *corpora albecantia* were seen. These appearances suggested that there had probably been an excess of follicle-stimulating hormone with diminution of luteinizing hormone. The follicles became almost mature, yet did not ovulate, and no *corpora lutea* were formed.

In answer to a letter of inquiry, the patient wrote on February 8, 1952, that she had had no child and had had no miscarriages since her operation. Up to October, 1951, her menstrual periods had been irregular, but since October they had been regular but painful. "I still have a few facial hairs but not near as bad as before operation. My blood pressure is still high and my weight is excessive."

Acknowledgements.

It is my pleasing duty to acknowledge my indebtedness to Dr. G. Davies and Dr. V. J. McGovern for their freely given assistance in the interpretation of the pathological features of the ovary, and to Mr. Woodward-Smith, of the Department of Medical Artistry, University of Sydney, for the illustrations which help so much in giving this report value.

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Legends to Illustrations.

FIGURE IV.—Photomicrograph of ovary, showing a large follicular cyst. There is hyperplasia of the granulosa layer. (Hæmatoxylin and eosin stain, × 75.)

FIGURE V.—A portion of the cyst illustrated in Figure IV showing luteinization of the *theca interna*. (Hæmatoxylin and eosin stain, × 300.)

FIGURE VI.—Photomicrograph of ovary, showing fibrosis of the stroma in which are lipoid histiocytes. (Hæmatoxylin and eosin stain, × 300.)

BICUSPID AORTIC VALVE ASSOCIATED WITH ANEURYSMAL DILATATION OF THE ASCENDING AORTA: REPORT OF A CASE.

By R. T. W. REID,

Department of Pathology, University of Adelaide.

Clinical Record.

A LABOURER, aged forty-nine years, was first discovered to have heart disease in 1944, when working with the Allied Works Council in the Northern Territory. He was referred to the Royal Adelaide Hospital with the diagnosis of *angina pectoris*, cardiac enlargement and aortic regurgitation. The angina was severe enough to warrant cervical sympathectomy in 1950, which, he said, improved him a little. He denied a previous rheumatic or syphilitic affection.

In October, 1950, full clinical investigation revealed unequal pupils, Corrigan's pulse, a to-and-fro murmur at the aortic area with a diastolic mitral murmur, and a blood pressure of 160 millimetres of mercury, systolic, and 60 millimetres, diastolic. A blood Wassermann test at this time yielded a negative result. X-ray screening of the chest in November, 1950, revealed a pulsatile aneurysm of the ascending aorta.

In May, 1951, the patient was readmitted to hospital after taking an overdose of barbitone, from which he recovered. Clinical examination suggested early *tabes dorsalis*, but lumbar puncture yielded cerebro-spinal fluid with a normal protein content, no cells, a normal colloidal gold curve and a negative Wassermann test result. Repeated blood Wassermann tests, and diagnostic Kline and Kahn tests yielded negative results. At this state bilateral inguinal hernias were repaired.

In November, 1951, he was readmitted to the Royal Adelaide Hospital because of severe pain in the chest, arms and right side of the neck. Breathlessness was now much worse, and he was noted to be coughing up heavily blood-stained sputum. The right lung base was dull to percussion, and examination of the cardio-vascular system showed aortic regurgitation. Death occurred five days later. It was thought possible that the aneurysm had eroded a bronchus.

Autopsy Findings.

Autopsy revealed the following relevant features.

The right pleural cavity contained a large amount of red-brown fluid, the pleural surface of the lung being reddened and shaggy. The right lung was collapsed, and the trachea and main bronchi were filled with a mixture of blood and froth. The middle and lower lobes of the right lung contained large areas of hæmorrhagic infarction, the vessels being plugged with ante-mortem thrombus. No venous thrombi were found in the leg veins.

The liver showed evidence of chronic passive venous congestion, and the brain and meninges were macroscopically normal.

The heart was grossly enlarged and together with the aorta weighed 800 grammes. The right atrium was dilated, the tricuspid valve admitting five fingers, and the right ventricular muscle was thickened. There was hypertrophy of the muscle of the left ventricle with dilatation of its cavity, but at the apex of the left ventricle the muscle was thinned out and mixed with white fibrous tissue. This area was covered by adherent mural thrombus. The leaves of the mitral cusp and its *chordæ tendineæ* were thin and, apart from lipoid deposits on the anterior mitral cusp, appeared normal. The coronary orifices were both patent; the left descending branch of the coronary artery showed atheroma of the intima and some localized calcified areas, but no occlusions were found along the artery's course. The right coronary artery was narrowed by atheroma 0·5 centimetre from the origin. The left atrium was dilated, its endocardium was normal, and the pulmonary valve was normal. The aortic

ring was widened, measuring 10 centimetres in circumference, and was bicuspid (Figure I). The bicuspid valve was formed by the fusion of the anterior and right posterior cusps, the site of fusion being still visible as a yellow ridge running from the attached border of the combined cusp to the aorta. The free edges of the single and conjoined cusps measured 5·0 and 5·5 centimetres in length respectively and the cusps were slightly thickened, especially along the edges. The ascending aorta was dilated, the dilatation commencing immediately above the aortic valve ring and ceasing at the origin of the innominate artery. The aneurysm measured 12·5 centimetres in diameter at the base of the commissures narrowing to 5·0 centimetres in diameter between the left common carotid and subclavian arteries. The intima of the aorta showed typical atheromatous plaques, but no scarring suggestive of syphilis.

Histological Findings.

Examination of sections of the left ventricle and left atrium showed muscle fibre hypertrophy. In the sections of the anterior wall of the left ventricle and its apex, fibrous tissue replacement of muscle with overlying organizing thrombus was seen. The aorta was sectioned just above the valve, and a strip was taken from this region running longitudinally up towards the aortic arch for a distance of 5·0 centimetres. The sections were stained with hæmatoxylin and eosin, Weigert's elastic tissue stain and the Hotchkiss-McManus periodic and leucofuchsin technique. The outer third of the media contained large blood vessels, whose walls were not thickened. The main mass of media was disrupted by spaces filled with granular material staining blue with hæmatoxylin (Figure II). The intima was slightly thickened in places by the fibrosis associated with atheroma. The adventitial vessels showed no periarteritis or endarteritis, but in an occasional one medial thickening was found. The elastic tissue stain (Weigert's) showed much loss of elastic fibres, and frayed fibre ends could be seen terminating at the edges of some cystic spaces (Figure III).

Discussion.

Several interesting features are apparent in the pathology of this heart.

Firstly, there is the presence of the bicuspid aorta. There is some doubt that the valvular lesion is one of the congenital variety, occurring as a result of fusion of the anterior and right posterior cusps. Evidence of the fusion is still apparent in the form of the ridge running from the conjoined cusp to the aortic root. These are the two cusps most commonly conjoined (Lewis and Grant, 1923) in the congenital variety. Gross (1937) suggests that bicuspid aortic valves occurring in adults should be considered congenital only if associated with other congenital malformations of the heart.

Secondly, there is the presence of the aneurysmal dilatation of the ascending aorta. Undoubtedly the commonest cause of aneurysm in this region is syphilis; this man was considered to have had clinical evidence of syphilitic aortitis, but on at least one occasion refused penicillin therapy. Macroscopically there is no evidence of syphilis in this aorta, nor is there other evidence of syphilis elsewhere in the body to support this. The Wassermann test results were negative in both blood and cerebro-spinal fluid. However, syphilis cannot be definitely excluded by a negative Wassermann test result.

The histological appearance of the aorta is interesting. There is gross destruction of elastic tissue with, in addition, material staining blue with hæmatoxylin between the collagen fibres and in small spaces. The material appears similar to that seen in Erdheim's cystic medionecrosis. Baer, Taussig and Oppenheimer (1943) describe two cases of what they claim to be congenital aneurysmal dilatation of the ascending aorta, associated with arachnodactyly, in which the histological appearance of the aorta is similar to this. However, in their cases the patients came to autopsy at the ages of fourteen and twenty-five years. Schorr, Braun and Wildman (1951) entertained the diagnosis in one case during life by angiocardiographic and clinical observations; the patient again was a young person. Harrison (1939) reported a case of coarctation of the aorta in a male subject, aged twenty-seven years, who at autopsy had a dilated ascending aorta; histological examination showed cystic areas in the media, some of which contained myxomatous material.

The interest in all these cases revolves around whether the association of the congenital heart lesions and coarctation with cystic degeneration is fortuitous, or whether the conditions are congenitally predetermined.

In this case, the patient's age (forty-nine years) is much greater than in the cases of Baer *et alii*. Furthermore, despite the absence of active syphilis, there is always the possibility that the aortic lesion may be the result of old syphilis. The balance of evidence, however, is considered to favour a non-syphilitic aortic aneurysm associated with a bicuspid aortic valve.

Summary.

A case is reported of bicuspid aortic valve lesion associated with a fusiform aneurysm of the ascending aorta.

Acknowledgements.

I wish to thank Dr. G. A. Lendon, honorary physician at the Royal Adelaide Hospital, for permission to publish the clinical record, and Professor J. S. Robertson, of the Department of Pathology, University of Adelaide, for helpful advice during compilation of the paper.

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Legends to Illustrations.

FIGURE I.—Photograph of aortic valve showing the two cusps; the conjoined one to the left was divided at autopsy.

FIGURE II.—Spaces in the media of the aorta. Granular material can be seen in the largest space. (Hæmatoxylin and eosin, × 320.)

FIGURE III.—Disruption of the elastic fibres in the media. (Weigert's elastic tissue, × 80.)

Reviews.

CLINICAL TROPICAL MEDICINE.

R. B. H. GRADWOHL and his fellow editors have chosen the 57 authors of their massive volume, "Clinical Tropical Medicine", almost exclusively from the Americas—North, Central and South.[1] This selection is responsible for both the best and the worst features of the book. For it is valuable as a reference work on tropical practice in the American region, and it is unsatisfactory as a treatise on tropical diseases as they occur throughout the world. This serious defect is disappointing in a costly book whose title and bulk should ensure a more comprehensive cover of its subject. Much of this criticism of the book would be obviated by altering its title to "American Clinical Tropical Medicine".

Dr. Gradwohl's difficult editorial task was made more arduous by the fact that most of the manuscript was submitted in foreign languages. An obvious flaw exists in his inequitable allotment of space to the various subjects. For example, African sleeping sickness (13 pages) occupies about the same space as fevers due to rat-bites (13 pages), and poisoning by scorpions and spiders (scorpionism and araneism) (14 pages). Yellow fever (17 pages) receives a poor deal when compared with *lymphogranuloma venereum* (25 pages), respiratory scleroma (20 pages) and snake-poisoning (called snake-venenation or ophidism) (27 pages). Relapsing fever is dismissed in five pages. While the American muco-cutaneous leishmaniasis is well done in 20 pages, kala-azar is inadequately dealt with in nine. Similar irregularity is shown in regard to references.

[1] "Clinical Tropical Medicine", edited by R. B. H. Gradwohl, M.D., Luis Benitez Soto, M.D., and Oscar Feisenfeld, M.D.; 1951. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 10" × 7", pp. 1670, with 473 illustrations and six colour plates. Price: £11 16s. 3d.

Considering the great importance of malaria in tropical practice, and the enormous accumulation of recent information on its various aspects, the sections on this disease are wholly inadequate. The subject of treatment, of which a proper appreciation is at present rendered difficult to practitioners by the amount of published material appearing, is covered in about three pages. A similar deficiency exists in the section on control measures, where the use of pyrethrum and paris green receive the same space as that of DDT. Blackwater fever is dealt with in "a few clinical points" in little over a page. In a work of this size, it is not enough to be told that blackwater fever is "not too common", or that a "navy clinician who spent more than a decade in malarial areas saw only three cases in nine years . . .". For it is when one of these rare cases suddenly appears that the practitioner, often far from consultative help, will turn to his reference book, and will reasonably expect some guidance in his procedure.

Despite the above criticisms, the book contains many excellent sections, and presents much of great interest to tropical clinicians. For the reasons mentioned, however, it is not recommended to Australian students, or to practitioners in the Australian tropical territories.

GYNÆCOLOGY.

J. P. GREENHILL's "Surgical Gynecology"[1] is an excellent companion volume for his "Office Gynecology". It is suitably condensed and, as the preface suggests, it was prepared for young gynæcologists and general practitioners, who also perform operations. Nearly all the gynæcological operations have been pictorially described in as simple a way as possible. The clinical aspect of gynæcology has been omitted, but an informative chapter on pre-operative preparation and post-operative care and complications is well done, particularly the discussion of water and electrolytic balance, early ambulation, shock, antibiotics and chemotherapy, and pulmonary and intestinal complications.

The description of operations for carcinoma of the vulva and cervix could have been omitted with advantage as they transgress the aims and usefulness of this book. Few gynæcologists would agree with the inadequate excision of a vulval growth shown, which would certainly invite a recurrence, nor with the method of lymph node dissection in the Wertheim's radical hysterectomy.

However, Greenhill's well-known clarity of description is greatly aided by the wealth of excellent illustrations of the operations described.

YELLOW FEVER.

"YELLOW FEVER", by Dr. George Strode and eight colleagues of the Rockefeller Foundation, is distinguished as the most comprehensive treatise yet published on this subject.[2] It is also a monument to the classical achievements of the International Health Division of the Foundation, of which Dr. Strode, the editor, is director.

In 1916 the Rockefeller Foundation established its Yellow Fever Commission, with the aim of eradicating yellow fever from the world. An epic attack on the problem was made from all sides by coordinated laboratory and field studies, supported by the vast resources of the Foundation. The cost of the project, to 1949, amounted to 14 million dollars, and the lives of six workers, who died of yellow fever. But though it was shown that the virus was entrenched in sylvatic haunts, and could not be eradicated, successful control measures were evolved. The achievements included the control of the urban mosquito vector, the discovery of an unexpected forest reservoir of the virus in monkeys and sometimes other animals, with transmission by bush mosquitoes, the development of protective vaccine, and the mouse protection test, which discloses the distribution of infection in populations. The extent to which the Foundation has identified itself with our present knowledge of yellow fever is indicated by the fact that this volume is largely based on the work of its own staff, and of agencies receiving its support and cooperation.

The book deals with both the preventive and the clinical aspects of yellow fever, including its transmission, pathology, epidemiology, immunology and clinical features. The writing is uniformly of high standard, and Dr. Strode is to be congratulated upon his editing. In all respects the volume is worthy of the great work it represents. It is excellently printed, well illustrated, and handsomely bound.

Although the areas of yellow fever endemicity are distant from Australia, the presence here of *Aëdes ægypti*, its urban vector, and the increasing risk of its introduction by air passengers, render the disease of more than academic importance to Australians. The book is essential for all health department and health laboratory libraries, as well as for medical libraries generally. As well, the wide biological implications of the subject should make it of interest to a large field of scientific workers.

CHRONIC BRONCHITIS.

"CHRONIC BRONCHITIS" is the result of Dr. T. H. Howell's research, the aim of which was to improve the management of the disease.[1] He gives briefly the views of past writers, and shows how common the condition is—11% of one series of patients attending general practitioners. There is a detailed account of the symptoms and signs, differential diagnosis and complications. Fifty-three patients were examined by *questionnaire*, but nothing new was discovered, and it is surprising that smoking is hardly mentioned in relation to the disease.

In 300 patients the morbid anatomy and cause of death are described, the commonest being right ventricular failure. A detailed bacteriological examination of the sputum in 50 cases showed the expected mixed and variable bacterial flora, which was only transiently modified by antibiotics.

Tested against coloured water, the expectorants were shown to be of no value, as their name would suggest, in promoting sputum production. The amount was extremely variable, 0 to 90 millilitres in twenty-four hours, and changes were more related to the weather than to cough mixtures. A similar experiment with sedative drugs showed equally little effect in the usual doses. There were no controls in the author's investigation of antispasmodic drugs, but he gives a clinical preference for aminophylline and "Benadryl" and considers these to be of value. It will be debated whether digoxin is contraindicated when the heart fails in this disease: the author prefers ouabain. He emphasizes the value of chest exercises and suppression of useless coughing; however, the disease remains one difficult to treat, and in this detailed account there is little that is new.

There are 32 illustrations, and a full bibliography at the conclusion of each chapter.

NEW WORLDS AND OLD.

IN a small volume of thirteen short essays, "New Worlds and Old", Professor Charles Singer has given us more of the fruits of his learning and long experience in a choice selection of biographical and philosophical dissertations.[2]

As might be anticipated in consideration of the ripe scholarship of this eminent medical historian, these few essays make delightful reading as we follow the evolution of scientific and religious ideas through the centuries; while considerable demands are made upon our powers of mental concentration if we take the trouble to reflect seriously upon the mighty impact of the "new philosophy" on a time-worn system of theology. However, those chapters likely to have a more ready and direct appeal by reason of their less weighty content deal with such subjects as Copernicus and Vesalius, the microscopic studies of Robert Hooke,

[1] "A Handbook of Operative Surgery: Surgical Gynecology: Including Important Obstetric Operations", by J. P. Greenhill, M.D.; 1952. Chicago: The Year Book Publishers, Incorporated. 8½" × 6", pp. 350, with 101 illustrations. Price: $8.50.

[2] "Yellow Fever", by George K. Strode, M.D., editor, and John C. Bugher, M.D., J. Austin Kerr, M.D., Hugh H. Smith, M.D., Kenneth C. Smithburn, M.D., Richard M. Taylor, M.D., Max Theiler, M.R.C.S., L.R.C.P., Andrew J. Warren, M.D., and Loring Whitman, M.D.; First Edition; 1951. New York: McGraw-Hill Book Company, Incorporated. 10" × 7½", pp. 726, with 77 illustrations. Price: $9.50.

[1] "Chronic Bronchitis", by Trevor H. Howell, M.R.C.P.Ed.; 1951. London: Butterworth and Company (Publishers), Limited. 8½" × 5", pp. 120, with two plates and 26 text figures. Price: 27s. 6d.

[2] "New Worlds and Old: Essays", by Charles Singer; 1951. London: William Heinemann (Medical Books), Limited. 7½" × 5", pp. 186. Price: 15s.

Aristotle as a naturalist, the botany of Theophrastus, and Galen as a modern. There is a fascinating account of the life and classical studies of the Scottish country practitioner, Francis Adams, still famed for his incomparable English translations of ancient Greek medical writings.

The six chapters interpolated between those of medico-historical import are philosophical and not so easy of assimilation. Here Professor Singer discourses eloquently on the various implications of the experimental method of scientific research as it has gradually impinged upon the traditional views of theologians and other authoritarians. If the reader is persistent and will keep on reminding himself that it is always harder to dig for gold than for potatoes, he is sure to find some reward in a deeper knowledge and clearer understanding of the problems involved in this seemingly impossible relationship. Professor Singer still affirms with some conviction that "religion has so vital a part to play that, without it, the continuance of civilised human society is unthinkable".

Nevertheless, there is a veiled suggestion in the next paragraph that the scientist may be able to get along fairly well with a kind of religion of his own, and these are the lines upon which his character and conduct may be shaped: "The sciences do, in fact, provide a way of life, a new way that would not have been understandable in former times. The effective prosecution of a science requires self-control and self-denial and strict regard for truth. Its pursuit needs courage and sensitiveness to the thoughts of others. Especially it demands, to a degree unapproached by any other discipline, extreme sensibility, both natural and acquired, to that wonderful form of beauty which is found in order. Further, on the plane of human relations, the first needs of a pursuit of a science are sober, industrious and modest living and, above all, loyalty to the great ideal of unflinching criticism of evidence, and therefore openness of mind, acceptance of correction, and outspokenness wherever error is in question."

It is no longer considered impolitic to discuss religious philosophies in the open forum; and it may yet lead to the discovery of still greater truths for the benefit of mankind.

SURGICAL TECHNIQUE.

"SURGICAL TECHNIQUE", by Stephen Power, is compact, but contains a considerable amount of valuable information concerning the minor technicalities of surgery which are usually overlooked or taken for granted in the standard text-books.[1] The steps of various surgical operations are not given, but that is not its purpose.

Uniformity in the choice of instruments is wisely stressed. Instead of the almost universal custom of assuming that the duties of an assistant at an operation are obvious to all, these are clearly given and the reasons for each explained. The principle of holding a row of interrupted sutures till they are all inserted and then cutting off the excess is illustrated. This is time-saving, but is practised more on the other side of the Atlantic than in Great Britain. The undesirability of taking uncovered blankets into the theatre is mentioned, but as this is a common fault it might have received more emphasis.

As this is essentially a book of instruction there is not the space in it for including many alternative, albeit equally effective, methods of doing things. A certain amount of dogmatism is thus unavoidable. However, tension sutures and rectal infusions, to mention only two points, have become less popular than is mentioned in the text. Non-chromicized catgut is condemned because of the variability in the rate of absorption, and yet it is concluded that "chromic gut is the stuff for all serious suturing" even though the time of its absorption is far from constant. Because of the frequency of complications following the use of silk sutures, there is a bias against other non-absorbable suture materials. It is stated that thread (presumably cotton and linen) "is open to the same objections as silk", whereas cotton and linen are both non-reacting cellulose, and silk is an animal protein. Further, cotton and linen do not unravel as silk does to form a network which will support and protect inflammatory granulation tissue.

In a book of this size much must be omitted, but brief reference at least might have been given to potassium deficiency and its treatment, to braided nylon as a suture material, and to "Zephiran", "Metaphen" and other commonly used skin antiseptics.

Technical details are well illustrated by photographs and drawings, the paper is good, and the type is very clear. This is a book for all resident medical officers interested enough to wish to learn the basis of surgical technique from a book instead of by trial and error.

[1] "Surgical Technique", by Stephen Power, M.D., F.R.C.S.; 1951. London: William Heinemann (Medical Books), Limited. 7¼" × 5", pp. 390, with 198 illustrations. Price: 30s.

DISEASE IN INFANCY AND CHILDHOOD.

PROFESSOR RICHARD ELLIS, occupant of the Edward Clark Chair in Child Life and Health in the University of Edinburgh, has written a new text-book, entitled "Disease in Infancy and Childhood", which outmodes all previous British works on pædiatric subjects.[1] It should be well known that tremendous advances have been made in the past twenty years in Great Britain in the study of the problems of infancy and childhood. The horizon has been considerably extended. It now includes every factor which influences, for weal or woe, the immature human being from conception to adulthood. The inclusion of new knowledge from this wider field of study and the blending of it with the older material are the features of main interest in this text-book.

We have become accustomed to expect good books from Edinburgh, and this one by Professor Ellis continues the tradition. Though comprehensive, the contents of this book have been purposely restricted to matters of clinical importance by the exclusion of medical curiosities. The author has shown great discrimination and teaching experience by grouping material according to degree of immaturity, embryological distortion, interference with function, reaction to a common stimulus and subsequent effect on the mature body. This unsystematic arrangement has minimized the necessity to include the same facts a number of times and has allowed the author to garner the grain for us already sifted from the chaff. He has earned our very warm thanks.

HANDBOOK OF SURGERY.

WHILE a student's text-book on surgery will occasionally enjoy considerable popularity in the absence of many illustrations, this is unlikely, no matter how excellent the text; and if "A Handbook of Surgery" by Reginald C. B. Ledlie and Michael Harmer[2] is to replace any of the standard surgical text-books it will probably need to have the number of illustrations greatly increased. Only 56 have been included, but those are excellent and are mainly line drawings.

Because of the desire to limit the size of the book to just over 500 pages and yet to mention most branches of surgery and surgical pathology, it has been necessary to present much of the text as a synopsis. This is an advantage for revision or reference; but it makes it rather difficult for the student commencing clinical work to read and assimilate its facts.

Brevity in some instances is almost carried to the extreme, for example, burns are dealt with in one and a half pages, and their treatment is very condensed. Against that, *spina bifida* receives the same amount of space. Few operative details are included, as is to be expected, but perhaps more indication could have been made of operative mortality and of post-operative complications. There is no doubt about the fact that "any nit-wit can diagnose advanced cancer", and it is pleasing to find that emphasis is laid on its early diagnosis.

Perhaps a little too much optimism is shown as to the results after depressed fractures of the tibial condyles. No indication is given of the difficulty that may be found in maintaining reduction. Lockhart-Mummery's operation for rectal prolapse of opening up the presacral space and packing with gauze is mentioned; but very few surgeons would

[1] "Disease in Infancy and Childhood", by Richard W. B. Ellis, O.B.E., M.A., M.D., F.R.C.P.; 1951. Edinburgh: E. and S. Livingstone, Limited. 10" × 6", pp. 704, with 300 illustrations, some of them coloured. Price: 42s.

[2] "A Handbook of Surgery", by Reginald C. H. Ledlie, M.B., B.S. (London), F.R.C.S. (England), and Michael Harmer, M.A., M.B., B.Chir. (Cantab.), F.R.C.S. (England); 1951. London: Baillière, Tindall and Cox. 8" × 5¼", pp. 542, with 56 text figures. Price: 21s.

agree that adhesions form to the posterior rectal wall so that "prolapse is prevented".

Despite these minor criticisms the book is well produced, the text is clear, and the price is reasonable. It is therefore recommended to the student as a summary of the essential facts which he will be called upon to know.

PHYSICAL DIAGNOSIS.

RAYMOND W. BRUST, of the University of Pennsylvania School of Medicine, has written a book which is well named "Physical Diagnosis", as the title describes the contents.[1] He places in antithesis history-taking with physical diagnosis, on the one hand, and special investigations, including laboratory tests and radiological examinations, on the other. In the introduction, Professor Schnabel punches the point home with a neat aphorism: "Physical diagnosis coupled with the patient's story constitutes the keystone to successful medical practice."

The book is a new one and the author claims originality only for the method of presentation of the facts. Though he has omitted information about pertinent special examination techniques and laboratory procedures, he is careful to state that the total evidence must be interpreted logically to reach a sound diagnosis.

With commendable clarity of diction, Dr. Brust describes the physical examination, by a master, of the several parts of the body in orderly array and then of the main systems, ending with an admirable exposition of the steps to be taken in the assessment of the functional integrity of the nervous system. Only where it is essential to the context has he allowed repetition of facts, and then he has done so intentionally to make cross-references unnecessary. The illustrations are workmanlike and sufficient without being intricate or ornate.

The use of such a book as this as a text-book for junior hospital students would be wise. As a refresher course for senior students or graduates in pursuit of higher degrees, the contents of this book would be extremely helpful.

BONE TUMOURS.

THE need for a good practical book on bone tumours has at last been supplied.[2] Most of the material in Lichtenstein's "Bone Tumors" will be familiar to those who have followed Lichtenstein and Jaffe's analyses of the material from the Hospital for Joint Diseases in New York. "Bone Tumors" deals with the clinical, radiological and pathological aspects of each subject followed by treatment and prognosis. Ingenious theories of histogenesis are avoided and the emphasis throughout is upon histological diagnosis. A sufficient number of cases of benign chondroblastoma, chondromyxoid fibroma and osteoid-osteoma have been observed for them to be regarded as entities, though it is doubtful if osteoid-osteoma is really a neoplasm.

Giant-cell tumour of bone is classified according to its activity and the prognostic value of histological grading is indicated.

It is pleasing to find a rational view on Ewing's tumour. When the author had excluded neuroblastomata, reticulum cell sarcomata, anaplastic secondary carcinomata and multiple myeloma from his material there was left a group which fell into a category of its own, that of Ewing's tumour.

A chapter on conditions which may be mistaken for bone tumours is rather scrappy.

The book is well produced and excellently illustrated by photographs of gross specimens, X-ray pictures and microscopic preparations. Table I on page 18 is not clear and furthermore, in it, "nonosteoblastic connective-tissue derivation" is applied to fibroma and giant cell tumour while "mesenchymal connective-tissue origin" is applied to Ewing's tumour. The distinction is not clear.

The book aims at conciseness. It succeeds, and is quite readable despite clumsy literary constructions. The verbs "to overdiagnose" and "to underdiagnose" recur rather frequently and there are other words and uses of words to which the purist would object. Notwithstanding, a copy of "Bone Tumors" should be on the desk of every pathologist, radiologist and orthopædic surgeon.

[1] "Physical Diagnosis", by Raymond W. Brust, A.B., M.D., F.A.C.P., with an introduction by Truman G. Schnabel, A.B., M.D., F.A.C.P.; 1951. New York: Appleton-Century-Crofts, Incorporated. 8½" × 6", pp. 306, with 60 illustrations. Price: $4.50.

[2] "Bone Tumours", by Louis Lichtenstein, M.D.; 1952. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 10" × 7", pp. 316, with 155 illustrations. Price: £5 10s. 3d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"After-Treatment: A Guide to General-Practitioners, House-Officers, Ward-Sisters and Dressers in the Care of Patients after Operation", by H. J. B. Atkins, D.M., M.Ch. (Oxon.), F.R.C.S. (England); Fourth Edition; 1952. 9" × 6", pp. 356, with 64 illustrations. Price: 30s.

The title is self-explanatory.

"Stereoencephalotomy: Thalamotomy and Related Procedures: Part I—Methods and Stereotaxic Atlas of the Human Brain", edited by E. A. Spiegel, M.D., and H. T. Wycis, M.D., F.A.C.S.; Volume I; 1952. New York: Grune and Stratton. 11" × 8½", pp. 184, with 79 illustrations. Price: $8.00.

Details of a neurosurgical method that the authors consider to be beyond the experimental stage.

"The Foot", by Norman C. Lake, M.D., M.S., D.Sc. (London), F.R.C.S. (England); Fourth Edition; 1952. London: Baillière, Tindall and Cox. 8½" × 6", pp. 472, with 166 illustrations. Price: 25s.

Completely revised, with much new matter added.

"Growth of Children: Sixty-Six Boys and Sixty Girls Each Measured at Three Days, and at One, Two, Three, Four and Five Years of Age", by Alexander Low, M.A., M.D., LL.D.; 1952. University of Aberdeen. 13½" × 8½", pp. 64. Price: 10s.

A record of measurements made in a stated way and by the same person upon the same children over a period of years.

"Surgery for Students of Nursing", by John Cairney, D.Sc., M.D., F.R.A.C.S., with a foreword by Miss M. I. Lamble, C.B.E.; 1952. Christchurch: N. M. Peryer, Limited. 9" × 6", pp. 334, with 120 text figures. Price: 40s.

The first text-book on surgical nursing to be published in New Zealand.

"The Medical Annual: A Year Book of Treatment and Practitioners' Index", edited by Henry Tidy, K.B.E., M.A., M.D. (Oxon.), F.R.C.P., and A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S.; seventieth year; 1952. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall, Limited. 8½" × 6", pp. 556, with 51 illustrations, a few in colour.

A hardy septuagenarian.

"Progress in Ophthalmology and Otolaryngology: A Quadrennial Review"; Part I—Ophthalmology, edited by Meyer Wiener, M.D., and A. Edward Maumenee, M.D.; Part II—Otolaryngology, edited by Percy E. Ireland, M.D., and Joseph A. Sullivan, M.B.; Volume I; 1952. New York: Grune and Stratton. 9" × 6½", pp. 680, with 19 illustrations. Price: $15.00.

A review covering the period from July, 1946, to the beginning of 1952.

"Manual of Electrocardiography", by Benjamin F. Smith, M.D.; 1952. New York: Elsevier Press, Incorporated. 9" × 6½", pp. 228, with 119 illustrations. Price: 32s.

An outline which starts with basic principles and, step by step, develops the theory of electrocardiography and applies it to the interpretation of electrocardiograms.

"Problems of Aging: Transactions of the Fourteenth Conference, September 7-8, 1951, St. Louis, Mo.", edited by Nathan W. Shock; 1952. New York: Josiah Macy Junior Foundation. 9½" × 6½", pp. 138, with nine text figures. Price: $3.00.

Contains papers and discussions on four subjects: biology and medicine; sociology, psychology, education and religion; economics, employment and welfare; medical services, hygiene and housing.

The Medical Journal of Australia

SATURDAY, NOVEMBER 1, 1952.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE AUSTRALIAN ABORIGINAL AND OURSELVES.

THE University of Sydney has recently announced the establishment of a University scholarship for a selected Australian aboriginal student. This is the result of a private benefaction (the sum of £200 *per annum* for three years is offered), but its acceptance by the University marks an interesting stage in the development of our attitude to the aboriginal peoples of Australia. The past history of this attitude is too well known to need detailed elaboration, but it is not, in general, to our credit. Writing in 1944,[1] the anthropologist, Professor A. P. Elkin, stated: "In 1788 there was a native population of from three to five hundred thousand, but now, after one hundred and fifty years of white occupation, there remain only about 60,000 full-blood aborigines." The causes of the decrease have been direct and indirect. Instances of deliberate brutality, though comparatively few, remain on record, but there is a certain dreary inevitability about most of the ill-effects resulting from contact between the aboriginal and the white invader—the clash of interests in relation to pasture lands and hunting grounds, with practically complete mutual lack of understanding of the other's point of view; the introduction of the white man's diseases and vices; the breaking up of tribal life with resultant social, economic and psychological instability. It is of little value to lament this now. In so far as we have been at fault as a people, we should seek to make amends to the aborigines who remain. It is useless to pretend that contact between whites and aborigines is not inevitable—few would regard the system of aboriginal reserves as the perfect answer—but the degree to which the harmful results of that contact can be mitigated or removed will depend largely on the intelligence and sympathy that we bring to bear on the problem. It would be much easier, of course, to let the aborigines die out, as the natives of Tasmania have long since done, but fortunately conscience and a sense of human responsibility still remain. The aborigines must be helped to adjust themselves to, and perhaps be assimilated into, our community and way of life, so that they may be in it and of it. As Professor Elkin puts it, "those who would save this people must, by action and word, help them to a fresh outlook on life and see that they are allotted to a definite part in Australian life".

At this point we come up against another problem—the monstrous fiction of racial superiority and inferiority. Most people repudiate this concept in its extreme forms, such as Hitler's anti-semitism and the more extravagant expressions of the colour bar; but to some degree it is taken for granted in the most surprising quarters. For this reason it needs to be actively combated, though it will not stand logical examination; it is at its best a fallacy, at its worst a vicious lie. Its most common expression is associated with skin colour. Whether we like it or not, this aspect is implicit in the term "White Australia Policy", and we must take the consequences while the term survives. Australia's immigration policy does not enter this discussion—it depends on economic and political factors not relevant to our subject and outside our province—but it is to be constantly reiterated that a "White Australia" concept has no justification from the scientific study of man. Just how the colour-bar idea has arisen is not at all clear. That it is comparatively modern was pointed out in a talk given some two years ago on the Third Programme of the British Broadcasting Corporation by an African medical man.[1] The speaker, R. B. Wellesley Cole, M.D., F.R.C.S., a West African from Sierra Leone practising in England, told of the travelling experiences of a High Court judge of his country (where there is no colour bar). All went well as far as Nigeria. In the Belgian Congo and Portuguese Angola, he and his wife were treated as "nothing but 'natives'". They had a wonderful time in Brazil and, with slight reservations, in the West Indies. In the Southern States of America they fared as might be expected. In New York they were received as visiting royalty in Harlem, but they never met a white American socially. We may hope that if they had come to Australia they would have found most people friendly and apparently unaware of their skin pigmentation; but they might have met some of the others. It would appear, as Cole states, that the colour bar attitude as we know it today arose about the seventeenth century; he cites, paradoxically enough, two potent factors in its development—the spread of Christianity and the rise of science. He pays generous tribute to great missionary work done by all sections of the Christian Church among non-Europeans and acknowledges the Christian role in the abolition of slavery, but is quite frank about this point on the debit side; indeed it is not difficult to see how more self-satisfied members of churches in Europe would react to the growing consciousness that the dark peoples of the East and elsewhere were "heathen" and so "inferior". The scientific factor was Linnæus's work on classification into genera and species, an unwarranted extension of which

[1] In an introduction to an illuminating collection of stories of aboriginal life in the Northern Territory entitled "Taboo", by W. E. Harvey (Australasian Publishing Company Proprietary, Limited, Sydney; Third Edition; 1944).

[1] *The Listener*, June 15, 1950.

(not by Linnæus) subdivided men. The basic factors in North America appear to have been economic, and the system became more or less crystallized; the Civil War, though it nominally enfranchized the American Negro, yet left him little better off. The South African position is described as the problem of an occupying people with a sense of superiority based on religious theories—a problem that has existed since the seventeenth century and has not yet been solved. In Brazil economic circumstances and a relatively low immigration rate have made adjustment and absorption of racial groups easier, and there is no colour bar. This brief account of Cole's views indicates how the colour bar has arisen, but it certainly does nothing to justify the bar. It cannot, of course, be justified, but it probably lies at the heart of most racial discrimination, and so arbitrarily and illogically opens the way for agitators and opportunists and all manner of strife.

It may be asked just what place this discussion has in a medical journal, but this is readily answered. Although the curriculum in most of our medical schools is circumscribed, the medical graduate is frequently expected to have, if not an authoritative, then a responsible opinion on many matters scientific, biological and anthropological that he would blush to claim as his own. However justified his diffidence, he has to admit that often in his immediate community no one else is better informed, though many may be less discriminating and more vocal. If he is asked his opinion on the inferiority of the Australian aboriginal he may ask if the grounds for inferiority are physical, intellectual, cultural, religious or what—and answer in his own mind with the thought of the tenor Harold Blair, the Australian Army officer Captain R. Saunders, the artist Albert Namatjira, the clergyman the Reverend James Noble and many more, realizing that no one has seriously suggested that these men are less representative of their people than the pathetic derelicts on the fringes of outback towns. If he is pressed to admit that in some other way, intrinsic but not defined, the aborigines are racially inferior, he may say, as Professor Elkin has said for the native peoples of the South-West Pacific,[1] that "there is no sound basis for any doctrine which would justify us in regarding and treating these peoples as inferior beings, whose wishes need not be respected and whose 'tribal' freedom is of no account". It is true, Professor Elkin goes on, that their stage of civilization is in some respects lower than ours; "but this fact simply puts on our shoulders the duty of helping them to develop that they may become full partners in the world's civilization". This, *mutatis mutandis* and with due humility towards people whose civilization is much older than ours, might well be applied in our world thinking. In our immediate sphere it will be interesting to see the outcome of the scholarship at the University of Sydney. It seems clear that, on past records, a suitable candidate should be readily available. Perhaps someone also will come forward to add to the amount offered, which, without any reflection on the generosity of the benefactor, could be supplemented with considerable advantage to the holder of the scholarship.

[1] "Wanted—A Charter for Native Peoples of the South-West Pacific", by A. P. Elkin (Australasian Publishing Company Proprietary, Limited, Sydney, 1943).

Current Comment.

HIGH-FREQUENCY BALLISTOCARDIOGRAPHY.

IN 1877 Gordon, applying Newton's theory of action equal to reaction, pointed out that there must be with each systole of the heart a footward reactive movement comparable with the impact of a rifle on the shoulder. Yandell Henderson (1905) was the first to record these movements. He used a table suspended on four long wires on which the subject lay and which was capable of free movement in a longitudinal direction, that is, head to feet but not sidewards. This method suffered from the defect that the appliance had a low frequency. After several attempts to improve on this technique, Starr in 1939 introduced a table also held by four wires and also capable only of movement in a longitudinal direction; but in this case he placed at one end of the table a strong steel spring which conferred high frequency and permitted small movements to be magnified and recorded. In a normal ballistogram, as Starr named it, four waves may be observed, named by him *H*, *I*, *J* and *K*. It is rather a reflection on the precision of the method that the *H* wave was, by one set of observers, referred to the "apex beat", by another to the contraction of the auricles. Some degree of unanimity is to be found in the ascription of the *K* wave to the resistance in the femoral arteries, for its reduction or abolition in cases of coarctation of the lower part of the abdominal aorta supports this view. A considerable literature grew up, particularly in America, on the use of Starr's technique in all sorts of conditions, physiological and pathological. Some criticism of the procedure, which really should have appeared at the outset, was made in 1945 by Hamilton and fellow workers, who showed that aortic pressure and elasticity affect the result, also that the ballistogram records only a portion of the recoil. Jongbloed (1951) improved the method by placing the table on four flat steel springs allowing only longitudinal movements, which were magnified optically and recorded photographically.

The whole subject of ballistocardiography has been examined with great thoroughness by A. Bouhuys,[1] who has proved experimentally that the movements can be profoundly influenced by adipose tissue and by posture. Hamilton's criticism he upholds, namely, that a portion only, and a variable portion, of the recoil is given by the apparatus. Bouhuys does not condemn the technique as useless; he states the opinion that with the same subject and with the same attendant conditions information of some value can be elicited. It is true that the electrocardiograph tells us nothing or next to nothing about the pumping efficiency of the heart, but we cannot accept the ballistocardiograph as filling this gap; indeed the history of this instrument is a warning against the undertaking of quantitative measurements before qualitative study has been carefully pursued and well established.

THROMBO-EMBOLISM IN MYOCARDIAL INFARCTION.

THE great interest that has been shown in the use of anticoagulant drugs in myocardial infarction has been based upon the realization that thrombus formation with its concomitant risk of embolism is one of the major risks of this condition. The anxiety felt by the physician that this complication may supervene is built on the mental image of the formation of a clot on the endocardium at the site of the lesion in the heart muscle. The difficulty is, of course, that there is really no means of knowing if and when this may occur, and if it does occur, what embolic sequels there may be. Therefore a study of thrombo-embolism has been undertaken by Robert A. Jordan, R. Drew Miller, Jesse E. Edwards and Robert L. Parker[2] with the object of adding to our knowledge of how

[1] *Proceedings, Koninklijke Nederlandse, Akademie van Wetenschappen*, Series C, Volume LV, March and April, 1952.

[2] *Circulation*, July, 1952.

often mural thrombus occurs in coronary occlusion, and how often it is followed by distant infarction. This study must perforce be confined to fatal cases, and it has been further limited by the authors to investigation of those patients who were not given any anticoagulant therapy during life. A clinical and pathological analysis has been made of 327 cases of acute and healed myocardial infarction; these include 210 consecutive cases of acute fatal infarction, in 110 of which there were healed lesions as well, and in addition to these 117 consecutive fatal cases were studied in which healed infarcts were discovered. The term "acute" is used in this series to mean infarcts which appear on clinical and histological evidence to have been formed within six weeks of death. As might be expected, most of the mural thrombi were found in the left ventricle (108 in number), while only nine were found in the right ventricle, and 31 in the auricular appendices. Special care was taken in the autopsies to collect evidence of other circulatory lesions, especially hypertension and congestive failure. The authors' findings have been tabulated so as to establish any legitimate connexion between the clinical phenomena and the anatomical findings. Mural thrombus in the left ventricle seems to be related to the following factors: the age of the infarct, its location, the presence of congestive cardiac failure and the presence of cardiac hypertrophy. Such thrombi were also found to be commoner in anterior infarctions than in posterior, though other factors may here be significant, such as the greater average size of anterior infarcts, and their greater degree of involvement of the apex of the heart. There are, of course, many such interdependent factors in such a study. Jordan and his associates conclude that the most important factors in the production of left ventricular thrombi are the presence of congestive failure and the presence of a large infarct. They also found that cardiac hypertrophy was a factor of importance. In brief, those conditions which tend to produce a relative degree of stasis in the circulation within the chambers of the heart are those favouring thrombus formation. This may seem to be a restatement of the obvious, but it is really more than this, being a restatement of the bases of correct clinical judgement in these cases.

In another article in the same journal Jordan and his colleagues have applied the same methods to a study of the factors concerned in arterial occlusion in the systemic and pulmonary circuits following acute and healed myocardial infarction. The symptoms associated with these arterial accidents are usually severe, and often ominous; further, they may arise soon after the cardiac accident, or after a variable and even considerable period of time. The same material was used as in the previous study. The prognosis of embolism is well shown in a diagram illustrating the lethal potentialities of the lesion in various sites. Thus, of 19 cases of embolism in the brain, five were fatal, and death occurred in association with embolism in the various sites as follows: lungs, in 13 out of 33 cases; kidneys, in none out of 27 cases; spleen, in none out of 17 cases; mesentery, in two out of 11 cases; extremities, in all of 13 cases. The patients so affected in this series were those who had suffered acute infarctions. Those who had post-mortem evidence of healed infarctions in the heart showed a somewhat similar distribution in the sites of distant occlusions by clot. Here, nine out of 20 were fatal in the brain, eight out of 25 in the lungs, and none out of twelve, four and two in the kidneys, spleen and mesentery respectively. The authors point out that venous thromboses apart from pulmonary thrombus are not included in their account, as these are outside the usual scope of an ordinary autopsy. They conclude that infarction of an organ or occlusion of a distant vessel may occur without the occurrence of mural thrombi in the left side of the heart, and emphasize that congestive cardiac failure following an acute infarct of the heart favours the occurrence of blocking of a systemic artery, and of pulmonary embolism. Healed infarctions of the heart presented certain differences, for the post-mortem material included cases in which surgical procedures had been carried out, or other medical conditions were present, making it difficult to decide the importance of pulmonary embolism or congestive failure.

Jordan and his colleagues remark that their inquiry might well be compared with a similar series in which anticoagulant therapy had been carried out and express the hope that such will be published. This should serve as a control, and should give a clearer idea of just how much can be expected from this form of treatment. However, their study has strengthened the basis on which rests the handling of patients after the occurrence of a myocardial infarction, even if, as is often the case, it is impracticable to use anticoagulants in a fully scientific manner, with adequate biochemical control.

MEDICAL CARE ABOARD AUSTRALIA-BOUND CONVICT SHIPS, 1786–1840.

STUDENTS of Australian medical and social history will be interested in an article by Donald R. McNeil, of Wisconsin, recently published in America.[1] The review is rather more an account of the evolution of the administrative aspects of health on convict ships and of the status of the ship's surgeon than a description of the medical problems themselves, although references to the latter abound. Using "Historical Records of New South Wales" and "Historical Records of Australia" as his chief sources, McNeil introduces his subject by drawing attention to the appallingly high incidence of illness in the Second and Third Fleets, even by comparison with the First, in which the convict mortality rate approached 6%. Apart from the conditions prevailing on the voyage itself, the embarkation of convicts already sick and the long periods of inactivity on board ship prior to departure contributed to this unsatisfactory state of affairs. During the terms of office of Governor Hunter and Governor King various attempts to improve the prisoners' lot were only partially successful. In 1814 Governor Macquarie requested and received a comprehensive report on the transportation system as it affected the health of the convicts from William Redfern, the Colony's Assistant Surgeon. Redfern, familiar to readers of "Out of the Past" in the columns of this journal, was well qualified to undertake the task, for he was an ex-convict, transported for taking part in the mutiny at the Nore while serving as a surgeon's mate. He advocated extensive reforms in the accommodation of prisoners, special attention being paid to the provision of washing facilities, adequate exercise and ventilation. Coinciding as it did with a reduction in British commitments in Europe, his plea for the appointment of competent naval surgeons to convict ships, in place of the newly qualified and incompetent doctors hitherto employed, could be granted. As a result Macquarie was able to report an immediate improvement in standards. Governor Macquarie also drew attention to other faults in the transportation system; in particular, he was largely responsible for the delegation to the ship's surgeon of the major share of the responsibility for convict health, together with the necessary authority to deal with it. Later surgeon superintendents not only carried out elaborate preventive measures and treated the sick, but also undertook to attempt the rehabilitation of the convicts by various means and to look after their spiritual welfare. However, as McNeil points out, these measures reduced the prisoners' discomfort, but did not greatly affect the strictly medical situation, improvement in which was limited by the medical knowledge of the period.

Much remains to be written of early Australian medical history, for the full significance of the role of disease—and of doctors—in the development of each of the Australian colonies has yet to be assessed. Papers presented by Dr. K. M. Bowden and Dr. B. C. Cohen at the recent congress in Melbourne indicate an increasing awareness of the work to be done in this field. The interest of an overseas observer cannot fail to serve as a stimulus and a challenge to Australian medical historians.

[1] *Bulletin of the History of Medicine*, March, 1952.

Abstracts from Medical Literature.

MEDICINE.

The Action of Dextran on the Clotting Mechanism.

A. B. LAURELL (*The Scandinavian Journal of Clinical and Laboratory Investigation*, 1951) records experiments showing that the addition of dextran in relatively low concentrations (0·2 to 2·0 parts *per centum*) to fibrinogen solutions or plasma shortens the clotting time determined after the addition of thrombin. Dextran also seems to have an inhibitory effect on the formation of thrombin. These two effects, it is pointed out, might well neutralize each other under physiological conditions, for the clinical experience is that dextran does not influence tendencies to hæmorrhage or thrombosis.

Geriatrics and the Physician's Outlook.

MEYER GOLDE (*The American Journal of Digestive Diseases*, October, 1951) discusses geriatrics and the problem of the physician's outlook on this subject. He records a number of case histories of old-age syndromes. He states that there has been in recent years an improvement in both the social and the medical outlook on the problem of age. He makes a plea for the eradication of the "old age" excuse for inadequate attention to illness among the elderly, and states that geriatrics should receive due recognition on a par with pædiatrics and that the subject should be included in medical school curricula. Retirement as now in vogue is over-mechanical and non-selective, with losses to professional life. The "old age" attitude on the part of general medicine should not go unchallenged.

Cortisone in Nephrosis.

M. RAPOPORT *et alii* (*The Journal of the American Medical Association*, November 17, 1951) describe the effect of ACTH on children with the nephrotic syndrome. They state that 34 children were studied. In all cases there was an insidious onset of œdema with albuminuria, low total serum content of protein and albumin, and high serum cholesterol level. ACTH can cause loss of œdema by diuresis in these children. Several courses of ACTH were given, 40 to 150 milligrammes daily. A dose of 50 milligrammes was quite effective, given at six-hourly intervals. Most children were treated for eight to twelve days before diuresis was produced. This might occur during treatment or up to four days later. The other signs of nephrosis mentioned also became less, as a rule, when diuresis was obtained. All the children did not respond to the treatment. The duration of the remission in these patients varied between one week and eighteen months. Of the 34 patients, 17 did not have a remission. The authors state that it is doubtful if the ultimate course of nephrosis was altered permanently by the use of ACTH. Many of the children showed humeral and metabolic changes due to stimulation of the adrenal gland by ACTH. Eosinopenia, weight gain and alkalosis with tetany occurred. For the last-named an amount of 1·5 grammes of potassium citrate was given daily during and after ACTH treatment until diuresis ceased. Cellulitis, peritonitis and septicæmia occurred in ten cases. When these first were observed penicillin or aureomycin was given prophylactically. Hypertension and tachycardia were noted in seven cases. Treatment for more than fourteen days or repeated courses at short intervals usually induced Cushing's syndrome. Moon face, acne, seborrhœa, hirsutism and cutaneous striæ were all observed. Transient glycosuria was detected in several cases.

Intermittent Drug Treatment of Pulmonary Tuberculosis.

F. J. HUGHES *et alii* (*Diseases of the Chest*, January, 1952) conclude from experiments upon 198 patients suffering from pulmonary tuberculosis, moderately or far advanced, that a dose of one or two grammes of streptomycin every third day combined with twelve grammes of para-aminosalicylic acid daily is the regimen of choice when specific drug therapy is indicated for non-miliary pulmonary tuberculosis for periods up to four months. A second form of combined intermittent therapy, employing both drugs every third day, was equally effective, but permitted the emergence of bacilli resistant to streptomycin. However, it was found advantageous for patients unable to tolerate daily administration of para-aminosalicylic acid, and it was more effective than daily treatment with streptomycin or para-aminosalicylic acid alone.

Aureomycin in Framboesia.

KENNETH R. HILL, KATERINA RHODES AND I. S. ESCOFFERY (*Transactions of the Royal Society of Tropical Medicine and Hygiene*, January, 1952) record the results of the administration of aureomycin to ten children suffering from framboesia in Jamaica. The children were aged from four years to sixteen years. Aureomycin was given in a dose of 25 milligrammes per kilogram of body weight per day for a period of fourteen days. The lesions were all early, and included primary lesions, papilliomata, papular lesions, condylomata and plantar lesions. The healing in the first weeks after the commencement of treatment appeared to be slower than when penicillin was used; but primary lesions healed in from one to six weeks, papillomatous and papular lesions within six weeks, condylomata within two weeks, and plantar lesions within six months. No relapse occurred during the period of observation (six months). Two patients failed to react to the Kahn test at the commencement of treatment and after six months. In five cases the titre of the Kahn reaction decreased greatly. In one the titre was the same after six months as at the commencement of treatment.

Tetanus.

R. S. DIAZ-RIVERA *et alii* (*The Journal of the American Medical Association*, December 22, 1951) discuss the effect of penicillin on tetanus *in vivo*. They state that 50,000 to 100,000 units of tetanus antitoxin given intravenously are enough to fix all the unfixed or free toxin in a patient with a chance of recovery. After this, 10,000 units should be given intramuscularly each day for four or five days. Sedation by intramuscular injection of "Sodium Luminal", and intravenous administration of dextrose saline (5%) for nutrition are necessary. Crystalline sodium penicillin G was given in doses of 30,000 units intramuscularly every three hours. The authors report 32 cases of tetanus. *Clostridium tetani* was grown in culture from suspected portals of entry in 17 cases. Penicillin was useless in the prevention of the diffusion or fixation of tetanus toxin; it helped to prevent and cure certain bacterial complications, especially pulmonary infections. The dose used by the authors was not limited. Streptomycin, however, was more useful when pneumonia developed, since Gram-negative bacteria usually caused the pneumonia. The *Clostridium tetani* disappeared in twenty-four hours up to twenty-nine days in the series reported.

Recovery from Diabetic Ketosis.

J. D. N. NABARRO, A. G. SPENCER AND J. M. STOWERS (*The Quarterly Journal of Medicine*, April, 1952) indicate what is involved in treating severe diabetic ketosis by presenting the results of metabolic studies on 19 of their patients. They bring out the following main points: (*a*) the enormity of the losses of water, electrolytes and cell protein, (*b*) the importance of eliminating glycosuria as quickly as possible, (*c*) the long time required for complete recovery. They state that recovery was quickest in regard to rehydration, for which four or five litres were retained in the first three days, but it took about ten days before the body's requirements of sodium, potassium and magnesium could be satisfied, and much longer, probably weeks, before protein balance was established. Adrenal cortical activity accounts for some of these changes, and as long as glycosuria is present excessive urinary losses of water and electrolytes occur, making replacement therapy more difficult. The importance of potassium balance is emphasized and the usefulness of special sodium chloride/lactate mixtures for intravenous therapy is made clear.

Occult Blood in Fæces.

C. D. NEEDHAM AND R. G. SIMPSON (*The Quarterly Journal of Medicine*, April, 1952) describe a very simple benzidine test for occult blood in the fæces. They state that the reliability of the test is not upset by the patient's continuing to take a normal diet (excluding large quantities of liver and black pudding) and iron-containing medicines. A positive result is given when three millilitres or more of blood are given by mouth.

Bronchial Asthma.

E. SCHWARTZ (*The Journal of the American Medical Association*, December 29, 1951) describes oral treatment of intractable asthma with cortisone. He states that daily doses were given until relief was obtained—300 milligrammes the first day, 200 milligrammes the second day, and 100 milligrammes daily thereafter. Oxygen therapy, intramuscular administration of epinephrine, and intravenous administration of aminophylline were continued during treatment with cortisone. For children 25% to 75% of the adult dose was used. When the asthma was relieved, reduced doses of cortisone were given. When cortisone was discontinued, asthma recurred, but it was often relieved by other methods. One patient was free of symptoms for two hundred and ten days after cortisone therapy. Low sodium content in the diet was used throughout. In

26 out of 31 courses of cortisone 22 patients gained relief. Other forms of treatment including allergy management were necessary.

A New Drug for Paroxysmal Tachycardia.

I. B. HANENSON *et alii* (*American Heart Journal*, February, 1952) describe the successful treatment of a patient suffering from recurrent ventricular tachycardia with procaine amide. The paroxysms were terminated by the intravenous injection of the drug, after which the patient received the drug orally each day for more than ten months without their reappearance, though they had previously occurred fairly frequently.

Treatment of Coronary Arterial Insufficiency by Implantation of Left Internal Mammary Artery into Myocardium.

A. VINEBERG (*The Journal of Thoracic Surgery*, January, 1952) has used the left internal mammary artery as a source of blood for the ischæmic myocardium in four patients suffering from crippling anginal pain. Preliminary animal experimentation showed that the artery is capable after implantation of nourishing the left ventricle. It does this by the formation of anastomoses with coronary vessels. In the ischæmic heart these anastomoses furnish a good blood supply to regions of the heart muscle formerly dependent on the partly occluded vessels. The operative procedure is short and simple and does not appear to embarrass the heart. Three of the four patients survived, one dying of coronary thrombosis before the desired anastomoses could develop. Two of the three survivors were freed from their pain.

A Cardiac Defibrillator.

G. L. BIRNBAUM (*The Journal of Thoracic Surgery*, February, 1952) describes a simple, portable and inexpensive electrical machine for use when ventricular fibrillation associated with cardiac arrest occurs in the operating theatre. This device, when two electrodes are placed one on each side of the heart, causes a current of 1·0 to 1·5 amperes to pass through the heart for approximately one-fifth of a second. The electric shock causes the heart to contract, and the heart beat may then restart. If the shock is followed by ventricular asystole, adrenaline is injected into the heart, which is then massaged. If ventricular fibrillation is resumed, procaine may be injected and further shocks administered. The author quotes G. S. Beck on the treatment of cardiac arrest as stating that "the beat can be restored in any normal heart". He states that no time should be wasted if there is no palpable pulse, no respiration and no blood pressure: the fourth or fifth left intercostal space must be incised at once, the pericardium exposed and the heart massaged, while the anæsthetist rhythmically and adequately inflates the lungs with oxygen. If the massage does not soon restart the heart, the pericardium is incised in order that it may be seen whether the ventricles are fibrillating or still. The *sine qua non* of success is boldly and quickly to open the chest when the indications occur. There are seven important things not to do: (i) Do not listen for a faint heart sound. (ii) Do not wait for an electrocardiogram. (iii) Do not inject adrenaline through the chest wall into the heart. (iv) Do not dilate the rectal sphincter. (v) Do not give artificial respiration by compressing the chest. (vi) Do not give a blood transfusion. (vii) Do not give an intraarterial transfusion.

Cortisone for Thrombocytopenia.

M. STEFANINI *et alii* (*The Journal of the American Medical Association*, June 14, 1952) report the effect of ACTH and cortisone therapy in cases of idiopathic thrombocytopenic purpura. Eleven patients were treated, eight cases being studied in detail. No permanent remissions were produced, but bleeding was lessened, the capillary fragility was reduced and the bleeding time was shortened. The authors consider that these drugs are of use in holding some cases in check till a spontaneous remission occurs, in some cases prior to splenectomy and in cases in which splenectomy has been unsuccessful.

Leptospiral Meningitis.

R. L. GAULD *et alii* (*The Journal of the American Medical Association*, May 17, 1952) draw attention to the importance of considering leptospiræ in cases of aseptic meningitis. They describe an outbreak among United States troops in Okinawa. The main features in their 16 cases were sudden onset of meningism and high fever, conjunctival congestion, muscular weakness and twitching, a normal white cell count and normal cerebro-spinal fluid at the onset. Fever lasted for six to eight days initially, and then a secondary fever lasting for two or three days occurred after a short remission. The cerebro-spinal fluid developed a high protein content and pleocytosis. There were no cases with jaundice and no deaths, and only one patient had residual weakness after three months.

Colitis and Pyorrhœa.

H. SENECA AND J. K. KARNIG (*The American Journal of Digestive Diseases*, May, 1952) have tested a suggestion that pyorrhœa and chronic intestinal disorders are frequently associated by referring 20 patients diagnosed as having *pyorrhœa alveolaris* for full medical examination. All but two had symptoms such as constipation, intermittent diarrhœa, abdominal pains, belching and so on; 14 were clinically suspected of having colitis; three proved to have amœbic colitis and seven streptococcic colitis. All had increased bacterial counts in the fæces. Treatment was mainly medical, the pyorrhœa disappearing as the intestinal disorder was cured.

Salt Depletion due to Chloramphenicol.

F. J. CATANZARO, A. M. OSTFELD AND G. E. BERGNER (*The Journal of the American Medical Association*, June 7, 1952) report a case of salt depletion from diarrhœa due to chloramphenicol. The patient was an elderly woman, admitted to hospital with anæmia and cardiac failure. She was given a diet of low salt content, digitalis, iron and vitamins. She had a single injection of an organic mercurial drug. Because of a raised temperature and leucocytosis, chloramphenicol therapy was commenced. Severe diarrhœa developed and caused moderate salt depletion. This was corrected, but death occurred. The anatomical findings at autopsy were deemed insufficient to explain the cause of death. It was considered that salt deficiency had contributed significantly.

Gastric Lesions in Löffler's Syndrome.

J. P. RUZIC *et alii* (*The Journal of the American Medical Association*, June 7, 1952) report a case of Löffler's syndrome with a transitory inflammatory lesion of the stomach simulating carcinoma. The patient had asthma, changing pulmonary shadows and pronounced eosinophilia. He then developed mild abdominal symptoms, and X-ray examination showed a large filling defect in the stomach involving the lesser curvature. At operation the stomach was found to be thickened, reddish brown and slightly nodular in this area. Biopsy showed a chronic inflammatory process in which eosinophile cells were prominent. Recovery was uneventful, and three months later the X-ray appearances of the stomach and chest were normal and there was no eosinophilia. The authors suggest extension of the concept of Löffler's syndrome to include all conditions in which there is involvement of tissues with a transient eosinophilic reaction as part of an allergic process.

B.C.G. Vaccine.

M. I. LEVINE (*Diseases of the Chest*, May, 1952) summarizes the deficiencies in our knowledge of B.C.G. vaccine as follows: (i) no method has been developed for stabilizing the potency of the vaccine; (ii) no method has been devised for preventing rapid decline in the number of viable organisms in the freshly prepared vaccine; (iii) there is variation in potency and in the number of organisms present in similar amounts of B.C.G. vaccine from different laboratories; (iv) the degree of immunity conferred by B.C.G. vaccine and the duration of this immunity are not as yet determined; (v) possible differences in the immunizing qualities of the vaccine in different racial groups are not adequately known; (vi) the best method of vaccination is still open to question. He states that B.C.G. vaccination should not be offered as a substitute for other anti-tuberculosis methods; also that considerable further knowledge is required before B.C.G. vaccination can be accepted as a general health measure in the United States of America and even before commercial laboratories may be licensed to produce the vaccine.

Defibrination of the Pleura in Empyema.

L. C. ROETTIG *et alii* (*Diseases of the Chest*, March, 1952) describe the use of pure crystalline trypsin in the treatment of empyema of the chest, both tuberculous and non-tuberculous. The enzyme is dissolved in buffered physiological solution and injected intrapleurally. It quickly removes the pyogenic membrane and decreases the viscosity of the fluid, and it often sterilizes the cavity as well. When the duration of the empyema has been less than six months, this treatment alone may be expected to allow the lung to expand without surgical decortication in more than half the cases. The daily inhalation of an aerosol of trypsin was found to render copious, tenacious, purulent sputum much less viscous.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

XL.

CORNS, CALLOSITIES AND FOOTWEAR.

THE title "Corns, Callosities and Footwear" suggests the common picture of a person endowed with inadequate muscles and an inefficient first metatarsal unit, which has been further weakened by overuse in housework, shopping and standing at cocktail parties. The syndrome of foot decompensation initiated in this way is completed by the use of "fashion shoes" of either court or single-strap type with narrow high heels and narrow soles. The misery resulting from foot capsize and associated corns and callosities will persist until woman desires and achieves emancipation from the salesmanship of the shoe and hosiery designers.

Pathology.

Corns and callosities are localized areas of hyperkeratosis produced in response to mechanical forces applied to the skin. The method of production of corns and callosities in feet may be more readily understood if one remembers the digital corns of violinists and spin bowlers and the palmar calluses of oarsmen and some workmen.

Callosities are caused by intermittent friction applied with varying degrees of pressure to a movable structure. Simple friction between the shoe and the skin may produce a painless callus which is physiological and non-inflammatory. Callus of a painful type is usually formed in response to intermittent friction applied with sufficient pressure to produce stresses on deeper structures resulting in inflammation of low-grade intensity. This pathological callus may be due to superficial rubbing, but more commonly it is caused by an internal frictional effect. This is produced by stresses of a shearing type which develop between the bone and the skin when tissues are rolled by an unduly mobile metatarsal bone. A callus may vary in thickness throughout its extent according to the degree of stress to which each part of the callus is subjected.

A typical corn consists of a localized area of callus having a core which is harder than the rest. The common corn is always preceded by callus formation. As pressure increases, the cells of the *stratum corneum* are wedged tightly together into a compact mass to form a hard core. This concentration is due to intermittent impact greatly in excess of that causing the surrounding callus. The cells are compressed between two points, which must be sufficiently fixed to allow of transmission of the forces. A true corn, therefore, presupposes structural or functional fixation of the part. Most "corns" on toes are really calluses, which will, of course, progress to corn formation if increasing forces act through the foot against its covering. Conversely, many "callosities" are plantar corns. Callosities are produced by friction, either internal or external, and they indicate undue mobility of either the shoe or part of a foot and may occur in barefooted races.

Corns are produced by intermittent fixed pressure and indicate more serious incongruity between a foot and its covering.

The sites of callosities and corns tell their own simple story. A corn beneath the interphalangeal joint of a great toe is due to excessive pressure at this joint because of limitation of dorsiflexion at the metatarso-phalangeal joint—that is, *hallux rigidus*. A callus on the tibial side of the same joint occurs if the great toe overlaps the sharp border of the insole, which is often hardened with shellac for durability.

More detail is learned by observing the lines and degrees of thickness of a callosity and the angle which the central induration of a corn assumes. One can read in the shape of these conditions the direction of the lines of force. A longitudinal ridge on a plantar callosity means that the underlying metatarsal is not moving as freely as the adjacent metatarsals.

In addition to the ordinary hard corns, there are soft corns, vascular corns, neuro-vascular corns and "seed corns". The pathology is essentially the same, except that of "seed corns", which are of unknown ætiology. These usually occur in people with dry skins and of advancing years, and do not always develop at pressure points. A seed corn consists of an encapsulated mixture of epithelial débris and cholesterol.

Diagnosis.

A plantar wart is the only common skin condition which is likely to be confused with corns and callosities. The symptomatology is identical. A wart is an infective papilloma with an excessive proliferation of the *stratum mucosum*. In callosities the papillary skin lines show no break in continuity. Papillary lines either circle around a wart or stop at its margin. Callus may overlie or surround a plantar wart. If the overlying callus is excised, the tips of the papillæ are seen beneath; the bed is either a moist, spongy, soft area or a dry lesion which is often punctured with tiny black or brown dots due to points of hæmorrhage from the papillæ. Corns and callosities always occur at pressure points. Warts may occur anywhere on the feet, but frequently they occur on the soles at the points of pressure, because these are the sites of contact with the causative virus when the subject is walking in bare feet.

Ætiology.

Corns and callosities are invariably secondary phenomena, and their diagnosis and treatment are fatuous unless the causes are sought for, found and treated. They occur in association with claw feet, club feet, and other congenital deformities, such as hammer toes and exostoses, also with deformities due to nervous diseases (for example, poliomyelitis), with malunited fractures and arthritis *et cetera*, but by far the most common condition in which they occur is in foot collapse of varying degrees.

An apparently normal foot will collapse if either the load is too great or the foot is too weak. Overload may be due to overweight, but it is usually due to maldistribution of the load produced by one or more of the following: high-heeled shoes, muscle weakness, bad mechanics and disease affecting the superstructure. Intrinsic defects in feet may be due to bad footwear, congenital weakness, disease or injury. The common causes of foot imbalance are an inefficient first metatarsal, overuse and bad footwear.

Commonly, decompensation begins in the forepart of the foot from functional insufficiency of the first metatarsal, which produces "flattening of the metatarsal arch" or "metatarsalgia". It may commence in the hindpart of the foot and produce valgoid, everted or pronated heel—the so-called "flattening of the long arch" or "flat feet".

Whatever the precipitating cause, and whatever part of the foot is first affected, unless relief is provided the whole foot will ultimately capsize. The usual sequence is that an inefficient first metatarsal ceases to bear its correct share of body weight. Functional insufficiency of the first metatarsal is basically congenital in origin, and it may be due to varus deformity, to hypermobility or shortness of the first metatarsal, to backward location of the sesamoids beneath the neck of this bone, and occasionally to supination deformity of all the metatarsals at the mid-tarsal joint. Compensation is provided for a while by overload of the second and third metatarsals. The forepart of the foot becomes supinated and splayed, and the hindpart of the foot may pronate or evert. As the capsize progresses, *metatarsus varus* and *hallux valgus* with bunion and superimposed corn develop in the incompetent first metatarsal unit. Owing to overstrain of the second metatarsal particularly, and to a lesser extent of the third and fourth metatarsals, callosities and later corns develop beneath the heads of these bones. The respective toes develop a "hammer toe" deformity. They become dorsiflexed at the metatarso-phalangeal joints and flexed at the interphalangeal joints, and are frequently dislocated dorsally onto the neck of the metatarsal. Callosities and corns develop over the dorsum of the protruding proximal interphalangeal joints, and corns occur around the nails and on the tips of the toes.

A fairly common late outcome is forward displacement of the fibro-fatty pads from beneath the metatarsal heads. As a result of the forward rolling movement of the metatarsal heads, an action which is greatly enhanced by wearing high-heeled shoes, the fatty pad is constantly heaped forward like dough before a rolling pin. Eventually this forward protrusion of the fatty pads becomes permanent. This may be a factor in the production of hammer toes, and is certainly a factor in their persistence. Not only are tissues distorted, but function is impaired.

Relationship to Footwear.

Examination of the wear marks and contour of an old shoe provides a record of the foot in action. It must be appreciated that not only does the shoe deflect the foot to

its own shape, but the foot also moulds the shoe. With the normal heel-toe gait and the usual slight roll from the outer side of heel to great toe at the push-off stage of the walking cycle, slightly increased wear would occur on the outer side of the back of the heel and beneath the first metatarsal head.

A study of the wear marks on the heel and sole, the shape of the upper and the position and angle of crease marks will sometimes enable one to detect decompensation before deformity has occurred.

In the civilized environment of hard pavements and prolonged standing *Homo sapiens* is still struggling to maintain the erect position. Whilst the female of the species is biologically superior to the male, she is physically inferior. She is handicapped by overload from excess subcutaneous fat and maldistribution of the load on the feet, because of flabby muscles, poor posture and wide pelvis. Mother is on her feet for longer hours than father, and in consequence her muscles and feet are subjected to more fatigue. Even when correctly shod, woman is more liable to foot collapse than man. It is quite true that feet which are intrinsically weak, and which have never been handicapped by wearing incorrect shoes—and in some cases have never even worn shoes—may collapse and be affected with callosities. It is also true that sound feet will stand moderate abuse from incorrect shoes and not capsize. However, the vast bulk of foot troubles, the aches and pains, bunions and ingrowing toenails, in addition to corns and callosities, are thrust upon us by the faults to be found in our footwear. Women suffer more than men in this regard, mainly because woman's foot apparel is more unnatural than man's. The court shoe does not provide adequate support. Furthermore, it is largely retained on madam's foot by undesirable antero-posterior pressure between the heel and toes in lieu of the usual side-to-side pressure and support provided by lace-up shoes. This harmful antero-posterior pressure, which is more easily obtained by wearing a shoe that is too short, leads to callus formation above the heel and contracted toes with corns. When we see a nicely polished great toenail peeping through a small hole in the very centre of a toecap we know that not only is the long flexor of the great toe seriously handicapped in its effort to enable the great toe to grip the ground, but both the long flexor and extensors are encouraged to act as bowstrings in the development of *hallux valgus*. Nature is further insulted by the use of narrow shoes with high, sloping and narrow heels. The martyred Saint Crispin must constantly turn in his grave because of the abominations created under his patronage. Nature designed the heel to bear most of the body weight. By the defilement of high and sloping heels proper load on the heel is not permitted. Toes are jammed into the front of the shoe and are distorted. High heels in particular cause overload on the metatarsal heads and produce a splay foot by further weakening the latest evolutionary acquisition in the production of a stable foot—namely, the approximation of the first metatarsal to the other metatarsals. If a heel higher than one and a half inches is worn it is impossible for a normal person to dorsiflex the ankle as is necessary in correct walking. In these circumstances ambulation is attempted by the "mincing gait". Inability to dorsiflex the ankle is overcome by flexion of the knees with inevitable flexion of the hips and compensatory increased lumbar curve, round shoulders and protruding neck. The step is short and the free swing of good healthy walking is missing. When this gait is used the joints cannot be fully extended and this results in strain from the head to the toes. If milady would only leave the enhancement of the natural curves of her limbs and body to the *couturiers* she would protect her daughter and herself from most of their foot troubles. If she ceased to wear narrow, high-heeled shoes, the mincing gait and the protruding buttocks might be missed. However, the grace of the tennis court and beach would then be seen in the street, and feet would be something beautiful to display.

If feet are weakened by either congenital or acquired intrinsic defects or overload *et cetera*, then the use of bad footwear will tilt the beam and cause the feet to capsize.

Prevention.

The development of corns and callosities is preventible, and in most cases this means the prevention of foot imbalance. The main problem is the provision of shoes and hosiery suitable for the individual. Shoes should fit the foot and hose should be slightly larger than the foot. Feet are no more standardized than faces; in fact each individual has two feet which are rarely similar in size and shape. When fitting shoes, the larger foot should, of course, be fitted.

We have a heavy responsibility to children in these matters. Even in these days of high prices they should not be asked to "grow into" shoes that are too large when purchased. Although young children require a new size about every two months, they are entitled to a correctly fitting shoe throughout.

Many manufacturers are making a sincere effort to provide correct shoes for children. Constant vigilance by medical men is necessary to interfere with the sale of bad shoes and to encourage the production of correct shoes.

Children outgrow their shoes and hose and do not complain. The cramping which results from incorrect footwear interferes with the development of the foot and deforms its malleable components. The greatest need at present is to prevent young girls from wearing bad foot apparel. The modern miss becomes style-conscious soon after being weaned. Unless fostered by a thoughtless parent, this style-consciousness is not converted to style worship until adolescence. It is a common practice for a girl to have either a white or black patent leather pair of shoes of semi-court type with a single strap across the instep for "best". These shoes do not provide adequate support. Whilst worn for "best" they do not do much harm; but ultimately the shoes with socks to match become tight, and they are then worn daily until worn out. This is an important cause of damage to girls' feet. Many adolescent girls are seen with foot collapse. Men's and boys' shoes are now on the whole quite satisfactory. Eve's peculiar belief that "sensible" is synonymous with "dowdy" is not without a grain of truth when applied to correct footwear. As she would prefer style to comfort, it is advisable to insist on a correct shoe for wear at home or whilst at work, and as a compromise a choice of footwear is allowed for social functions. However, pregnant women should not be permitted the luxury of fashion. A convalescent patient should not be allowed to ruin the feet by the use of bedroom slippers, as these do not provide the firm support which is so necessary when muscles have been weakened by illness.

Bad mechanics or disease which weaken the superstructure or alter the line of weight-bearing should be borne in mind, the possibility of foot collapse considered, and efforts made to prevent it. Many structural and postural defects should be recognized and treated in infancy and early childhood; for example, the use of heels wedged and elongated on the inner side will protect the feet from the undue strain imposed by knock-knees, bow-legs, tibial torsion and congenital defects in the hindpart of the foot, such as *talipes calcaneo-valgus*.

People whose occupation entails prolonged standing (which causes more strain on feet than prolonged walking) should be permitted regular rest intervals. They should also be taught to rotate hips laterally to reduce strain on the inner side of the feet and to adopt the position of "attention" or "at ease" from time to time. Foot hygiene and comfort are improved by changing footwear during the day or by the use of two pairs of light socks which are reversed at midday. Shoes retain their shape and the insole does not buckle up if shoe trees are used and the shoe cupboard is located in a cool room.

Correct Footwear.

The best material for socks and stockings is pure silk or light-weight wool. Both of these materials are poor conductors of heat and readily absorb moisture. Ideally, there should be right and left socks. The hose worn should be a little larger than the foot. Shrinking socks and growing feet are a potent cause of deflected toes and contracted toes in childhood. Most women know that nylon stockings make their feet sore, but they prefer "eye appeal" to comfort. Nylon, whilst it wears well, is hard and does not absorb sweat.

If the foot is functioning properly and is in a well-fitting shoe they should work together as nearly as possible as a unit.

Figures I and II and the specifications were published in this journal in 1950, in a letter from Dr. A. R. Hamilton, honorary secretary of the Australian Orthopædic Association (Hamilton, 1950). The specifications there state that a correctly made shoe should have a flat, broad heel (A) and be laced with a high quarter (B). The stiffener (C) in the heel should be firm and high and extended forwards on the inner side to the level of the scaphoid tubercle (D) and to an equal distance on the outer side, so as to grip and control securely the child's foot. The width of the shoe across the toes (E) should be equal to that across the metatarsal heads (F). There must be ample height at the toe end of the shoe (G), so that the digits are not forced into a confined space—to this end a "block toe" stiffener is advocated. There must also be adequate depth in the shoe for the metatarsal heads (H). The length between that portion of the shoe housing the first metatarsal head and the toe tips (I) must be in correct proportion to the total

length of the shoe. A new shoe should have approximately five-eighths of an inch clearance beyond the extremity of the longest toe, and all measurements should be made whilst the child's weight is being borne on the feet. The sole of the shoe must be sufficiently rigid to preserve its shape. The general outline of the shoe should be either (i) inswung, for most feet, or (ii) straight along its inner border. The shank of the shoe will be either (*a*) waisted (J) or (*b*) broad (K)—depending upon the type of foot to be shod. An old saying of Bespoke Bootmakers states: "Look after the great toe when fitting shoes and the rest of the foot will look after itself." Whilst the specifications refer to children, they are suitable for adults, with the following amplifications. The insole of a shoe should be the same size and shape as the sole of the foot when bearing weight. The shoe, whether machine-made or hand-sewn, is built around this component. Both rigid and flexible footwear are allowable. For general use most people need a shoe with a rigid shank which retains the arch of the shoe. This is provided by a steel

FIGURE I.

shank which lies between the insole and outsole and extends forward from the heel to a point immediately behind the tread. The sole or outsole should be thick, varying from one-quarter to three-quarters of an inch, according to the subject's age, sex and occupation. Heavy soles act as insulation and have more resiliency than the pavement. Thin soles do not provide adequate protection, and are only for infants or feeble old ladies who rarely walk. The sole should be flat and not of the rocker type. Welt soles are to be preferred to either pump soles or the fashionable stuck-on soles, as they are more durable. The person's heel should fit snugly into the heel "quarter" of the shoe. This should be asymmetrical in cross-section and bulge postero-

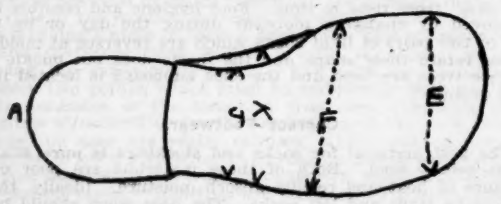

FIGURE II.

laterally to a slight degree in order to accommodate the *tendo Achillis* insertion. It should also be lower on the outer side to avoid rub on the lateral malleolus. The primary concern is that the anterior part of the foot be fitted. The adjustment of a loose heel is a secondary consideration. This may be corrected by means of a felt lining or a celluloid heel cuff, or on occasions by having the heel taken in.

The vamp should be of adequate width and depth and be slightly more anatomically correct than the foot. When a "hump" is present over the first cuneiform region great care is necessary to select and fit a shoe which will not cause pressure. Pressure over this bony thickening at the first cuneiform metatarsal joint may be relieved by having a bootmaker stretch the shoe, by the use of recessed felt padding at the base of the tongue, by wearing boots, or by the doubtful expedient of wearing a court shoe.

Fitting of Shoes.

Fitting of shoes under X-ray control is mainly good salesmanship. With brief exposures it is not harmful. It is of value in the circumvention of the cheerful schemer, aged two years, who coils his sensitive toes when touched. Without X-ray evidence it may be difficult to determine whether the toes have been coiled up on purpose or whether the vamp is not high enough to allow them to lie naturally. When the desirable hard toecaps or toe puffs are present, it is often difficult for a parent to feel the tips of the toes, and in these cases an X-ray view is helpful to reveal the position of the bones.

Correct fitting of footwear is the responsibility of the individual or parent. A trained shoe fitter, when available, is invaluable; but frequently the shop assistant is only a salesman who may have gravitated from meat-packing. The fitting is different. People generally wear a fitting which is too narrow. Expert shoe fitters state that the average woman feels insulted if asked to wear a shoe the insole of which is the same size as her sole. By the provision of shoes with soles so narrow that she must walk partially on the upper, designers of fashion shoes foster this delusion of *"très petit pied"*. Temporary relief from foot strain is often secured by the use of a narrow fitting. The foot is then supported by the overlapping upper. This extra support disappears when the upper stretches from weight-bearing for which it is not designed. These shoes are then discarded to swell the number of slightly used shoes in the cupboard. When the foot constantly overlaps the sole or heel, shearing stresses on the edge of the insole produce calluses on the inner border of the great toe and on both sides of the heel.

Shoes should be functional, and so should fit and support the feet whilst leaving the greatest possible degree of freedom in the use of muscles which control foot movement. Some feet depart from the standard norm and can scarcely ever be comfortably fitted to a ready-made shoe. Variations in the length of the first metatarsal bring the "great toe joint" either too far to the front or too far to the rear to fit the line of most ready-made lasts, and in these cases shoes should be made to measure. Long, narrow feet are difficult to fit, as are also short, squat feet. The natural make-up of people and their feet is variable, but they conform to type. At the end of the scale are the above-mentioned two groups. One type of subject is the loose-limbed individual who possesses undue laxity of ligaments and atonia of muscles. As he is so dependent on muscle control, the foot as a stable lever is likely to be reduced in value if the control is inefficient, and functional footwear is necessary to prevent collapse. The other type is a short, stocky, muscle-bound individual. He walks with a jerky gait without normal flexibility. Short steps are taken with absence of elasticity, and so weight-bearing points take excess pressure.

For many patients with gross foot deformity due to congenital defect, trauma or nervous disease it is necessary to have shoes made to measure. For most people with functional foot imbalance the use of a ready-made shoe of correct design will be satisfactory. When capsize leads to moderate deformity a shoe can be altered to fit the foot. A correct shoe can be stretched by a bootmaker to accommodate particular deformities. In lieu of or in addition to this, a small metal boss can be attached to a stock wooden tree at the position corresponding to the deformity, and the shoe can be stretched by this tree. If the degree of deformity is so pronounced that this stretching will not allow accommodation of the deformity, then pressure over the deformed toes can usually be relieved by the use of "balloons" or "gussets" in the upper of the shoe. The leather of the vamp is removed at the site of pressure by the deformed digit and a piece of looser and thinner leather is stuck over the hole in the upper. If this is done carefully the alteration is invisible. The lining is stretched but not cut.

Treatment.

The essential treatment of corns and callosities is, of course, the provision of suitable footwear and the treatment of the cause, whether it is either a congenital or an acquired deformity.

The local treatment of corns and callosities affords real but temporary relief from pain and can be undertaken by the patient or with more safety by a trained chiropodist. A person with diabetes or peripheral vascular disease cannot afford the economy of self-pedicure. These patients should be under the care of an adequately trained chiropodist for supervision of foot hygiene and treatment of nails, corns *et cetera*.

The bulk of a callosity may be reduced by rubbing it with pumice stone or paring it with a knife after previous softening by the application of a 20% salicylic acid ointment. Any paring should be done only after the skin has been cleansed and softened further in a bath of warm, soapy water. Corns should be removed not only to relieve pain, but also to restore elasticity to the skin. They may either be removed by the use of a salicylic acid corn paste or a

plaster, or enucleated with a knife. The painful soft corns which commonly occur between the fourth and fifth toes usually require excision. If these corns recur it may be necessary for a surgeon to excise the underlying osteophyte which is usually present at the base of the fourth toe. Protective pads of adhesive chiropody felt bevelled at the edges may be used to relieve pressure. The limit to the use and design of these pads is that of the ingenuity and skill of the doctor or chiropodist.

Fat pads may be wasted as the result of injury, disease or degeneration. Their shock-absorbing function is best replaced by the use of sponge rubber stuck over heels and rigid joints, and by wool felt over mobile parts. Rubber is not suitable for use over mobile parts, as it is elastic in all directions. This causes it to drag in the opposite direction to the movements of the foot. The cushioning effect of rubber and felt pads is soon lost by pressure, and therefore constant renewal is necessary. When plantar corns and callosities occur in this type of patient cushioning is provided and pressure avoided by the use of inner soles made of cork, of lamb's wool or of rubber with leather covering and recessed over the points of maximum pressure.

Functional foot capsize is treated in the following manner. When collapse is slight it may be necessary only to provide a correct shoe and instruct the patient in foot and correct walking exercises. Women who have worn high heels habitually often require the *tendo Achillis* to be stretched gradually by exercises and by only gradually lowering the height of the heel. In all cases remedial measures are taken to correct posture. When collapse of moderate degree occurs it is essential to force the first metatarsal bone to take its fair share of the body weight. This may be actively accomplished by the use of the Hauser metatarsal bar, which forces the anterior part of the foot into relative pronation. A Hauser bar is an inclined plane higher on the outer side than on the inner side and so the weight is forced onto the big toe. The ability of this bar to force the inefficient first metatarsal to bear weight is increased by the use of a felt pad with a similar inclined plane directed medially and placed beneath the lining on the insole at the level of the metatarsal necks. An alternative method of forcing the first metatarsal to bear weight is to provide packing beneath the metatarsal head. This is achieved by the use of a "dome" beneath the metatarsal necks with a "platform" extending further forward beneath the first metatarso-phalangeal joint. The latter is a passive and supportive method, which is often more successful in the treatment of older people.

Reeducation to reestablish normal gait is essential, as the use of either type of shoe alteration for many years will not prevent the recurrence of symptoms. Correct walking is a heel-toe gait, in which there is an even roll from the outer side of the heel to the great toe.

Surgery is used as an adjunct in treatment to correct *hallux valgus, metatarsus varus,* contracted toes, osteophytes and forward displacement of the plantar fat pads. By these measures function is improved and deformity corrected so that a ready-made shoe can be worn in comfort.

Conclusions.

Corns and callosities may occur in a person with foot deformity when correctly designed shoes are worn. Conversely, a person with good feet may develop corns if bad footwear is worn. In general, however, there is the triad of bad footwear, foot collapse, corns and callosities.

Under civilized conditions the foot is not called on to function naturally. It is a weak organ which can be strengthened in most children by games played in bare feet on soft ground. However, many children, because of overweight, knock-knees, valgoid feet *et cetera,* need the support of shoes at all times.

Most persons require shoes with rigid shanks to satisfy the needs of a civilized existence, even at the sacrifice of some of the natural function of the foot. A shoe with a straight inner border is ugly and clumsy, but skilful internal deviation at the waist can, to some extent, conceal this.

It is a matter of some social importance that an artistic type of foot apparel be constructed to embody the principles of correct footwear, and so diminish the large numbers of mature women who are now left like roués to regret the indiscretions of their youth.

Acknowledgement.

I wish to thank the Australian Orthopædic Association for permission to reproduce Figures I and II and the specifications.

L. J. WOODLAND,
Sydney.

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British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held at the Repatriation General Hospital, Springbank, South Australia, on June 19, 1952, the President, DR. R. L. THOROLD GRANT, in the chair. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staff of the hospital.[1]

Multiple Myelomatosis.

DR. R. V. SOUTHCOTT presented a patient suffering from multiple myelomatosis. The patient, a man, aged forty-five years, had been admitted on May 22, 1952, to the psychiatric ward, complaining of restlessness, depression, irritability, malaise, insomnia *et cetera.* A routine chest X-ray examination was made, and the radiologist reported osteolytic deposits in the ribs and clavicles, suggestive of secondary carcinomatous deposits, or possibly multiple myelomatosis. The patient was transferred to a medical ward for further investigation. His history was that he had had the nervous symptoms mentioned for the past three years, but he thought that his present illness had started about eighteen months before his admission to hospital. The first symptom was that he noticed pain in his hips on odd days, sometimes on one side, and sometimes on the other. The pain would last for only one or two days, then clear up for months. On account of the pains in his hips, he found that he had to be careful when lifting things. By occupation he was a motor repairer, and had previously been a leading hand in charge of tramway vehicle repairing. The only noxious substance to which he had been exposed was petrol vapour, and that to a less extent than any bowser attendant. In February, 1952, the pain finally settled in the left hip, being fairly constant, occasionally increasing, but never leaving him. If he tried to pick up a heavy object, for example, his toolbox, he would find that he could not move, and would have to call one of his work-mates to get the toolbox out of his hand so that he could move. Afterwards he would be all right again, so long as he was careful. Then in March, 1952, he had begun to get a pain deep in the bones, he thought, about the middle of his left forearm. He found that he could not hold a fork at the table properly, but there had since been some improvement, so that over the past two months he had been able to lift books with his left hand. In May he had begun to feel an aching in his right shoulder. A pain started in his right temporal region, and ran down the side of his face into his shoulder. That was the worst ache of all, and had been present constantly since, as had also an ache in his right collarbone. He thought that his right collarbone had swollen. Also on three or four occasions there had been a severe ache below his right scapula. Since being in hospital he had developed an aching in the ribs on the right side of his chest. He also had an ache in the calves, relieved by hot water bags. There had been no cough. He had neck stiffness only when his face and neck ached. He had suffered for years from headaches and constipation, and there had been no recent change in those symptoms. His past history included infection with measles, diphtheria, mumps and whooping-cough and appendicectomy. His family history contained nothing of significance, both parents having lived to over the age of eighty years.

On examination he looked pale and ill. General weakness was found around his shoulders, especially when the arms were abducted beyond the horizontal position. There were no tender spots in the skull. No tenderness was elicited on the sternum, but some tenderness of the right upper anterior region of his thoracic cage was present. There were some tender spots at the time of examination. Tenderness was elicited on pressure over the spines of the second and sixth thoracic vertebræ. Examination of his heart showed no abnormality. The blood pressure was 145 millimetres of mercury, systolic, and 90 millimetres, diastolic. There was some flattening of the percussion note at his lung bases,

[1] Acknowledgement is made to the Chairman of the Repatriation Commission for permission to publish the details of these cases.

but otherwise no abnormal physical sign was detected. Examination of the abdomen and genitalia showed no abnormality. There was some tenderness to pressure in his calves. Neurological examination revealed no significant abnormality. On rectal examination the prostate felt normal. Further X-ray examinations were made, and these showed punched-out osteolytic areas in the skull, spine, shoulder and pelvic girdles *et cetera*. The left articulation of the first cervical vertebra with the occiput was destroyed. Severe bone destruction was present in the body of the eleventh thoracic vertebra, but no collapse. Several ribs showed severe destruction, as did both ilia and the sacrum. The femora, humeri, radii and ulnæ all showed the typical punched-out areas, which varied in size from about two millimetres up to, in the femora, areas shown in the X-ray film measuring 44 by 26 millimetres; in the left radius there was an extensive region of destruction just below the head and three inches (7·5 centimetres) long. There was no radiological evidence of any lesions in the tibiæ and fibulæ. Biochemical investigations of the blood gave the following findings: the serum calcium content was 12·5 milligrammes per 100 millilitres (normal 9·0 to 11·5 milligrammes); the serum inorganic phosphorus content was 5·3 milligrammes per 100 millilitres (normal 2·0 to 5·0 milligrammes); the serum alkaline phosphatase content was 8·4 King-Armstrong units per 100 millilitres (normal 3·0 to 13·0 units); the serum acid phosphatase content was 0·5 Gutman units per 100 millilitres (normal 0·5 to 5·0 units); the blood uric acid content was 6·2 milligrammes per 100 millilitres. The urine contained no evidence of Bence-Jones proteose. The hæmoglobin value of the blood was 10·9 grammes per 100 millilitres. The white cells numbered 7200 per cubic millimetre, and a differential white cell count showed that 84% were neutrophile cells, 12% were lymphocytes, 3% were monocytes and 1% were eosinophile cells. The mean corpuscular volume, mean corpuscular hæmoglobin, and mean corpuscular hæmoglobin concentration were all normal. The total protein content of the blood was 8·6%—2·4% albumin and 6·2% globulin; the albumin-globulin ratio was 0·4. Dr. J. A. Bonnin reported that the sternal marrow showed heavy rouleau formation, which was a usual finding on marrow examination in myelomatosis, especially when the globulin contents were increased. The sternal marrow examination showed 23% of myeloma cells. The myeloid-erythroid ratio was 6·1 to 1·0 (normal, 3·0 or 5·0 to 1·0). On microscopic examination the marrow was less cellular than normal with suppression of the erythroid tissue. A large number of myeloma cells were present, which were large and immature and had prominent nucleoli. Binucleate giant cell forms were relatively common, but cells with three or more nuclei were rare. The cells were not well-differentiated plasma cells, but also were not extremely anaplastic.

Dr. Southcott went on to discuss the treatment of multiple myelomatosis. He said that the prognosis in that disease was poor, and various forms of chemical and irradiation therapy had been tried with variable results. There was no one mode of treatment which was commonly accepted as being the best. Probably urethane had been as widely used in the past as any drug, with varying results. He quoted the work of Rundles and Reeves (1950), who had stated that with urethane therapy clinical improvement occurred in the first three months, with, however, little radiological improvement. After four to six months usually recalcification occurred. The sooner treatment was instituted the better. Another form of treatment commonly used was radiotherapy, and recently Lawrence and Wassermann (1950) had studied patients treated with radioactive phosphorus (P^{32}) or radioactive strontium (Sr^{89}). Dr. Southcott quoted those authors as stating their opinion that the use of the radioactive substances mentioned was better in some ways than radiotherapy, and as suggesting a form of treatment with one of those radioactive materials together with the diamidene compounds and urethane. He quoted other work in which the newer derivatives of nitrogen mustard were being used—for example, compound "9500" or trisethylene-imoino-S-triazene. However, he stressed the importance of not neglecting general measures such as blood transfusion and treatment by iron and diet.

DR. J. A. BONNIN discussed the hæmatological features in the case. He pointed out the importance of sternal marrow puncture in those cases, it being virtually diagnostic. He referred to the studies of Baird on the disease, and considered that, from the fact that the myeloma cells were not well-differentiated plasma cells, nor extremely anaplastic, the clinical course of the disease in the case shown should be of moderate severity and duration only. The prognosis, according to Baird, depended on the degree of anaplasia of the myeloma cell, and in Dr. Bonnin's experience at the Royal Adelaide Hospital, the patients in whom the myeloma cells were not well differentiated towards plasma cells responded better to urethane therapy when that drug was tolerated.

DR. M. E. CHINNER discussed the treatment of the disease. He said that he had recently had under his care a patient with myelomatosis who had not responded at all to urethane therapy. He thought that in some cases attempts to prolong the patient's life by treatment only added to the burden of his suffering. His recent patient had had his life made a misery by the multiple fractures of his ribs.

DR. D. A. HICKS discussed the differential diagnosis from parathyroid tumours, pointing out the importance of urinary calcium estimations, as well as blood levels, as diagnosis, and said that accurate estimations of urinary calcium excretion were difficult to obtain. He referred to a recent case of his of recurrent renal calculi, typical of parathyroid tumour, with normal blood calcium and phosphorus levels.

Fracture-Dislocation of the Cervical Part of the Spine.

DR. G. C. THORNTON presented a patient who had sustained a fracture-dislocation of the cervical part of the spine at the levels of the third and fourth cervical vertebræ. The patient, a serving member of the army, was admitted to hospital on February 2, 1952, quadriplegic from the injury. Tidal drainage of the bladder, intravenous infusion and duodenal suction were carried out in the first forty-eight hours for urinary retention and abdominal distension. Massive collapse of the left lung occurred on February 6, relieved by tracheal aspiration. Crutchfield's tractor pulled out on the sixth day, and was replaced by a tractor of Blackburn type. There was a reappearance of a flicker of movement and sensation of the right side of the body at the end of forty-eight hours, indicative of the partial nature of the cord transection. With his recovery from the spinal shock came return of normal intestinal peristalsis, and oral feeding was commenced on the ninth day. In five weeks the patient showed a return of normal voluntary control of the bladder, the residual infection having responded to specific chemotherapy. The patient had no memory for the first four weeks of his stay in hospital, and he was noisy, disorientated and fearful of night, and showed no response to sedation. A diagnosis of *dementia tremens* was made, and the patient became placid and uncomplaining when given half an ounce of brandy four-hourly and no other sedation. There followed a complete return of power on the right side of the body, with scattered return, much behind that of the right, on the left side. The traction was discontinued in twelve weeks, with return to the original deformity of the cervical part of the spine. At the time of the meeting, eighteen weeks after the injury, the patient was able to walk with the aid of crutches; weakness of the left hand and tendency to extensor spasms of the left leg were his main disabilities, and he was commencing to complain of root irritation. Dr. Thornton advocated some form of external fixation, for example, tibial grafts and/or wiring, early in recovery, with the provision of a brace to shorten the bedridden state, and stressed the importance of the value of early relief of the respiratory embarrassment, often a terminal event, by tracheotomy.

DR. N. S. GUNNING mentioned the difficulty of "holding" the cervical part of the spine in correct alignment, and paid tribute to the nursing staff on the care of the patient.

DR. WILSON spoke on the inadequacy of Crutchfield's "ice-tongs" for prolonged traction, and said that he had seen so many cervical spines "concertina" to their original state of deformity, with little subsequent disability, that a major surgical procedure was not warranted.

Intramedullary Pegging for Ununited Fracture of the Femur.

Dr. Thornton's second patient was a Korean veteran, who had undergone intramedullary pegging of an ununited fracture of the femur. Four inches of overlapping were overcome, and the patient was weight-bearing in a calliper three months after the insertion of the peg, with good knee movements and normal new bone formation. Dr. Thornton said that he considered that the ideal method in the absence of sepsis.

Aneurysm of the Internal Carotid Artery.

DR. R. V. SOUTHCOTT showed a patient with an aneurysm of the internal carotid artery. The patient, a carpenter, aged thirty-six years, had been admitted to the hospital on May 3, 1952. He had previously been given the diagnosis of anxiety hysteria. Over the past six months he had been

working very hard and doing a good deal of overtime. He had begun to be nervy from this, and to experience restless nights and nightmares. About four weeks prior to his first admission to hospital, on April 10, a family argument precipitated a "nervous breakdown" with weeping on his part, requiring the attention of his local doctor for sedation. Over the few days following the emotional upset, he experienced a constant dull ache in his right frontal region. That had persisted, but he had gone about his work. A fortnight later, on May 24, he was standing on a stool in a house being built by his employers in Adelaide, holding a lightweight batten against the roof with one hand, and marking off its length with a pencil in the other hand. He said that there was no strain involved in that. He suddenly felt a terrific pain in his forehead, right behind the eye itself, which he described as "like a thousand knives piercing into the back of the eye". He fell off the stool, dropping the batten, and sat on the floor holding his head in his hands in an attempt to relieve the pain. There was no loss of consciousness. After about a quarter of an hour the pain "dulled off", and he was able to get up and walk around, and was driven home by his friends, when he went to bed. The "terrific pain" disappeared entirely, and he was left with the "constant dull ache" previously described, which, however, was rather worse than before. His right eyelid began to droop about a week prior to his admission to hospital. In about four hours his upper eyelid had drooped right down, and his eye had closed. He noticed an intensified headache at the onset of the ptosis. He had experience of some pain in the back of the head also. He was a heavy smoker, using one ounce of tobacco per day. He drank alcohol moderately, two pints of beer per day. As a child he had suffered from measles and whooping-cough. During his army service he had suffered from malaria, dengue, and a gunshot wound of his left shoulder, as well as a rash following a prophylactic injection against tetanus. At the age of twenty-six years, while still in the army, he had undergone tonsillectomy.

On examination, the patient was seen to be a tall, thin and excitable man, frequently tearful. An obvious right ptosis was present. His right eye was abducted. His right pupil was larger than the left then and it remained so. On his first admission to hospital, a slight reaction of the pupil to light and accommodation was recorded. At the time of the meeting there was no reaction to either light or attempts at accommodation. His left pupil reacted normally to light and accommodation. Although his right eye remained constantly abducted, a small amount of movement inwards and rotation inwards could be controlled by the patient. His ocular signs had changed very little over the last six weeks, and indicated a complete lesion of the right third nerve, the fourth and sixth cranial nerves remaining intact. Some weeks prior to the meeting it was recorded that his right eye was proptosed, and there was tenderness over the distribution of the right supratrochlear nerve. That was no longer in evidence. There had been no papilloedema, but there had been some impairment of right conjunctival sensation. Thus, apart from the involvement of the right third cranial nerve and the ophthalmic division of the right trigeminal nerve, no neurological abnormality had been found. There had been no venous dilatation around his orbits, and no intracranial bruit was to be heard. Over his chin there was a Y-shaped congenital cleft, which filled with hair, and which he could not shave properly. No abnormality had been noticed in his cardio-vascular or respiratory symptoms. The blood pressure remained at 135 millimetres of mercury, systolic, and 90 millimetres, diastolic.

The patient was kept at rest with the aid of sedatives. He was emotional and frequently wept, and on occasions complained bitterly of pain in his occipital region, which was relieved almost instantly by any proprietary acetylsalicylic acid tablet.

A lumbar puncture was performed on May 6. The cerebrospinal fluid was clear and colourless, and microscopic examination revealed no cells. The pressure was 280 millimetres of water. The patient was subsequently treated with magnesium sulphate retention enemas, and there had been no further development of significance and no improvement. Lumbar puncture had not been repeated.

Dr. Southcott said that it was considered that the history and findings indicated an aneurysm in association with his right internal carotid artery, causing a complete lesion of his right oculomotor nerve, and with some irritation of the ophthalmic division of his right trigeminal nerve, in the region of the cavernous sinus. Presumably some lesion had commenced with his initial headaches, but a much grosser lesion had occurred with the knife-like pains behind the right eye. There could not have been a gross lasting communication with his cavernous sinus, otherwise more signs of venous engorgement would have been in evidence. Similarly, it was thought that no gross subarachnoid hæmorrhage was likely to have occurred, otherwise there should still have been some evidence of blood staining and cellular reaction in his cerebro-spinal fluid. Over the two days preceding the meeting there had been some reduction in the ptosis on the right side, and he was able to lift his right upper eyelid a little. A right carotid angiogram, taken recently by Dr. P. W. Verco, confirmed the presence of an aneurysm of the internal carotid artery.

Dr. Southcott discussed the symptomatology and differential diagnosis of the disease. He said that cerebral tumours were the most important condition requiring to be differentiated, and laid stress on the difference in the speeds of onset in the two diseases—the one appeared with dramatic suddenness, and the other had a comparatively slow onset. He discussed the ætiology under three headings: (i) mycotic aneurysms (4% in Dandy's series), (ii) aneurysms secondary to arteriosclerosis (16%), (iii) aneurysms from congenital weaknesses usually at arterial junctions around the circle of Willis (80%). As there was no evidence of arteriosclerosis or hypertension in the case under discussion, and no preceding disease likely to cause a mycotic aneurysm, there was little doubt that the diagnosis belonged to the third and commonest category of congenital aneurysms.

Discussing treatment, Dr. Southcott said that the treatment of choice was surgical, and that whereas the mortality rate with conservative treatment was in the region of 50%, with surgical intervention the mortality rate had been reduced to the neighbourhood of 15%. Dandy's operation and ligation of the carotid artery intracranially and extracranially were mentioned, as well as the work of Jefferson and others. The importance of pre-operative procedures was stressed.

DR. P. W. VERCO demonstrated the radiological features of the angiogram that he had taken in the case. Owing to the extreme nervousness on the part of the patient during the procedure, filling of the carotid vascular system with radio-opaque materials was not so good as expected, but none the less, an aneurysm of the right internal carotid artery was clearly shown, just medial to the proximal intracavernous sinus part of the artery.

DR. M. E. CHINNER discussed the pathological features of the disease, stressing the importance of clotting in relation to the aneurysm, and of second hæmorrhages. Dr. Chinner advocated ligation of the right common carotid artery in the case under discussion, as this would reduce the circulation through the aneurysm by about one-third, and thereby reduce the chance of second hæmorrhages.

Radiological and Other Features of the Bronchopneumonias.

Dr. Southcott next showed the X-ray pictures from a series of patients with bronchopneumonia treated at the hospital over the last two years. He discussed the clinical histories of a number of the patients, and pointed out that the bronchopneumonia seen in elderly patients admitted to the Repatriation General Hospital usually occurred in previous sufferers from bronchitis, asthma, emphysema or else pulmonary fibrosis secondary to old injuries or inflammatory processes. Several such cases were discussed and X-ray films were presented. Dr. Southcott went on to say that on the other hand, in the younger group it was frequently possible to allot the case of "bronchopneumonia" to some ætiological group.

Discussing diagnosis, Dr. Southcott said that considerable help was required from the radiological department and the laboratory. He attempted to carry out the following regime after clinical history-taking and examination: (i) X-ray examination as soon as possible, a postero-anterior film and a lateral film of the appropriate side being taken; (ii) culture of the sputum; (iii) a differential white cell count and hæmoglobin estimation; (iv) a Weil-Felix test, repeated later; (v) preservation of serum for later testing for complement-fixation against "Q" fever and psittacosis; tests for cold agglutinins were also carried out. Dr. Southcott said that these tests were done on both an initial specimen of serum and a later specimen. By these tests it was possible to differentiate these cases of pneumonia into ætiological groups with considerable success.

Dr. Southcott next discussed two cases with the diagnosis "primary atypical pneumonia", using the term to cover a coherent group and excluding pneumonias from known viruses. One of these patients was a war widow's child, aged twelve years, who had been admitted to hospital on January 12, 1951, with pyrexia, frequent unproductive cough, splenomegaly and enlarged lymph glands. Examination of

the lungs gave negative results. The pyrexia disappeared after one day's treatment with aureomycin. The X-ray films of the chest showed an opaque area at the base of the right lung, which had decreased considerably by January 22, and disappeared by January 26. A second patient was a serving member of the navy, aged twenty-four years, who had been admitted to the hospital on March 15, 1952, as possibly suffering from malaria, with a history of a rigor and sweating on the morning of his admission. Cough was his most prominent symptom, and he had some headache, drowsiness and backache. He was febrile, but otherwise physical examination revealed no abnormality. An X-ray examination of his chest had shown "a little lessened translucency of the right upper zone". The only positive laboratory finding was a cold agglutinin titre of approximately one in 30. Dr. Southcott said that neither of the last two patients showed evidence of other ætiological factors, and they were referred to as suffering from "primary atypical pneumonia".

Psittacosis.

Dr. Southcott next showed a patient suffering from psittacosis. The patient, a man, aged fifty years, had been admitted to the hospital on August 13, 1951, complaining chiefly of a backache which he had had for eight weeks, and which he ascribed to shovelling gravel. He had suffered a gunshot wound of his back in 1918. He had also on a past occasion been diagnosed as suffering from neurasthenia. The condition of his back had improved over the last two weeks, and he had been able to travel from Western Australia to South Australia.

On his arrival in South Australia he had gone to live in an unhygienic house in Goodwood. The previous owners had kept many fowls and a parrot. The patient said that there were "bird droppings everywhere", and he had had a dusty time sweeping up the droppings. For a few days prior to his admission to hospital, he had been "shivery and shaky", and had developed a dull headache with stabbing pain in the head. He had an unproductive cough and some shortness of breath. He complained also of "icy waves" when in bed. He had a temperature of 99° F. on the evening of August 18, and of 98·8° F. on the morning of August 22, but otherwise his temperature was not elevated, and his pulse rate was between 72 and 90 beats per minute. Physical examination of his chest revealed no abnormality, and no concern was felt for his respiratory system until the routine chest X-ray examination made on August 16 showed an area of infiltration in the upper zone which aroused suspicion of active tuberculosis. There was some evidence of old right-sided pleurisy, which presumably dated back to 1917, when he had gone overseas at the age of sixteen years, and suffered right-sided pneumonia with pleurisy, and a hæmoptysis later in the year.

The investigations were unrevealing, and a report on a further chest X-ray examination on August 30 was to the effect that the opaque area previously reported in the upper zone of the left lung had now almost completely resolved, and there was no evidence to suggest active tuberculosis. No treatment by antibiotics had been instituted. The patient was discharged from hospital, well, on September 12, 1951, and had remained well since. The Weil-Felix and Paul-Bunnell tests both gave negative results; the Mantoux test gave a positive result (second strength tuberculin); six specimens of sputum were tested for acid-fast bacilli, with negative results; sputum culture for acid-fast bacilli was carried out with negative results, only a non-specific flora being grown on ordinary culture. The psittacosis complement-fixation titre was one in 560, which was very high and diagnostic of psittacosis. The history of contact with bird droppings also was confirmatory. The titre of the patient's serum had gradually dropped until at the time of the meeting it was one in 32. The complement-fixation tests were carried out by Dr. J. A. R. Miles of the Institute of Medical and Veterinary Science, Adelaide, and the patient had since provided serum for testing for psittacosis. The patient had had mild psittacosis and had recovered without antibiotics.

Dr. Southcott then said that the only other case of psittacosis found by Dr. Miles in the last couple of years in Adelaide had also been in a patient under his care. A man, aged fifty-two years, had been admitted to hospital with recurrent pneumonia in September, 1951. His complement-fixation titre for psittacosis was one in 16, indicating an old infection. Subsequent estimations of his psittacosis complement-fixation titre showed that it remained at one in 16. There was no recent history of contact with birds, but he had a history of pneumonia whilst he was a prisoner-of-war in Burma in 1943, and a more suggestive recent history of pyrexia with severe headache that had developed after he had been working on a fowl-house in 1948. No chest X-ray examinations were made during the 1948 illness. It was possible that the 1948 "pyrexia of unknown origin" could have been due to psittacosis.

Dr. Southcott went on to say that it was usually stated in North America that one case in 10 of viral type pneumonia was psittacosis; in South Australia the incidence appeared to be much less.

Pneumonia and Endemic Typhus.

The next X-ray films presented were those of a policeman, aged forty-six years, who had been admitted to the hospital on January 14, 1951, with a history of a shivering attack five days previously; he had a continuous frontal headache, severe sweats, and weakness with a pyrexia of three days' duration, one of the recorded temperatures being 105° F. On the evening of his admission to hospital he complained of a severe pain in his left side. He had no cough of any significance, and no glandular enlargement. He was sent in for investigation as possibly suffering from malaria. Although he had not been in a malarious area for five years, he had suffered from attacks which had been diagnosed as malaria about once every twelve months for the previous five years, without blood examination, and had been treated with "Atebrin". The condition of his lungs was recorded as clinically normal prior to his admission to hospital.

On his admission he was cyanosed, feverish and distressed. His tongue was furred. The right side of his chest moved normally, but respiratory movement of his chest was diminished on the left side. The percussion note was dull at the base of the left lung, and fine râles were present there, as well as pectoriloquy above it. The heart sounds were soft and rapid. His blood pressure was 165 millimetres of mercury, systolic, and 100 millimetres, diastolic. There was some generalized abdominal rigidity, but no viscus was palpable. He was admitted to hospital with the diagnosis of left basal lobar pneumonia, and treated with morphine, aspirin and penicillin, 200,000 units given every four hours. Examination of a blood film failed to reveal any malaria parasites. The hæmoglobin value was 15·5 grammes per 100 millilitres and the leucocyte count was 6900 per cubic millimetre, the following differential count being 58% neutrophile cells, 32% lymphocytes, 6% monocytes, 3% eosinophile cells and 1% basophile cells.

An X-ray examination of the chest was performed on January 15, and the report of the radiologist was: "The heart is enlarged in its transverse diameter. The lung markings are prominent at each lung base, and no localized consolidation was detected."

On January 16 the patient felt rather better. He was still cyanosed and restless. The percussion note at his lung bases was dull, on the left more so than on the right. Coarse râles were present on inspiration at each lung base, and ægophony and pectoriloquy were present at the base of the left lung, and vocal resonance was unimpaired at the base of his right lung. By January 17 he had not responded to the penicillin therapy, and this was changed to a course of "Chloromycetin", 500 milligrammes being given three hourly for eighteen hours, then 500 milligrammes six-hourly. With this therapy he improved slowly, his pyrexia diminished, and his lung signs lessened, until therapy was stopped on January 24. At this stage he was apyrexial. Physical examination revealed only a few fine râles at the lung bases, mainly at the left. Sputum culture on January 22 had shown a growth of *Streptococcus viridans*. There was no glandular enlargement or splenomegaly at any stage. The Weil-Felix titre on January 26 was one in 2560 to Proteus OX19, being less than one in 40 to Proteus OX2 and OXK. On January 30 the test was repeated; the OX19 titre was one in 5210, and the other titres showed no significant alteration. He had begun to have another rise of temperature, and a course of aureomycin was instituted, 500 milligrammes being given six-hourly. There were a few râles to be heard at the base of his right lung. The chest X-ray examination of February 1 was reported as showing no appreciable alteration. By February 20 his Proteus OX19 titre was one in 640.

Dr. Southcott said that the case was interesting in showing an intermediate type pneumonia in a case of murine typhus, with radiological findings somewhat different from clinical expectation, a recrudescence of pyrexia after cessation of "Chloromycetin" therapy which was sometimes described, and some unusual clinical features in the absence of lymphadenopathy and splenomegaly. Confirmatory epidemiological evidence was given in this case by the

patient's statement that in the course of his police duties he frequently used to visit Warradale camp, which he described as being heavily rat-infested.

Other Virus Pneumonias.

Dr. Southcott discussed the pneumonias which he said occurred occasionally in various well-known virus infections. He showed the X-ray films from a case of chickenpox pneumonia in a man, aged twenty-three years. This patient had also had encephalitis. Both the pneumonia and the encephalitis had cleared up quickly after the commencement of aureomycin therapy, as had occasionally been reported in the literature for chickenpox pneumonia. Dr. Southcott said that the mortality rate for that disease in the period before the antibiotics was high, and he intended to report the case more fully elsewhere. Pneumonia from measles was also mentioned. Dr. Southcott said that he had not had a case of "Q" fever diagnosed in the hospital over the last two years, and discussed the radiological features. In conclusion, he pointed out how difficult it was to make a correct ætiological diagnosis in a case of pneumonia at the present time.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

AN ACT CONSEQUENT ON THE ACT OF JUNE, 1838.[1]

Medical Qualifications Act.
2 Vic No 22.

Whereas an Act was passed in the present Session of the Legislative Council intituled "An Act to provide for the attendance of Medical Witnesses at Coroners' Inquests and Inquiries held before Justices of the Peace", wherein it was, amongst other things, enacted That whenever upon the summoning or holding of any Coroner's Inquest, or the holding of any Inquiry before a Justice of the Peace, it should appear to the Coroner that the deceased person was not at, or immediately before his or her death, attended by any legally qualified Medical Practitioner then it should be lawful for such Coroner, or Justice, or Justices as the case may be to issue a summons for the attendance as a witness at such Inquest of such legally qualified Medical Practitioner in actual practice as should reside nearest to the place where such Inquest was holden and whereas it has thereby become necessary to declare who shall for the purposes of that Act be deemed a "legally qualified Medical Practitioner".

Be it therefore enacted by His Excellency the Governor of New South Wales with the advice of the Legislative Council thereof That no person from & after the first day of January next shall for the purposes of the said recited Act be deemed a legally qualified Medical Practitioner unless such person shall have proved to the satisfaction of the President, and any other Member of a Medical Board, to be hereafter appointed, that he is a Doctor or Bachelor of Medicine of some University or a Physician or Surgeon licensed or admitted as such by some College of Physicians or Surgeons in Great Britain, or Ireland, or a member of the Company of Apothecaries of London, or who is, or has been, a medical officer duly appointed and confirmed of Her Majestys sea or land Service.

2. And be it further enacted, That it shall, and may be lawful for the Governor, or Acting Governor for the time being, to appoint a Committee consisting of not less than three Members, being of the Medical Profession, one of whom shall be nominated President, together with a Secretary under the style and description of "The New South Wales Medical Board", and it shall be lawful for the said Governor, or Acting Governor for the time being to remove the said Members or any of them, and upon the removal, death, or resignation of the said members or any of them, to appoint such other person or persons as he shall think fit and any person desirous of being declared a legally qualified Medical Practitioner as aforesaid shall submit his degree diploma or other certificate or proof of his being so duly qualified for the examination and approval of the said Medical Board and shall obtain from the said Medical Board a certificate of his being so qualified.

3. And be it further enacted That the said Medical Board shall, on or before the first day of January next cause the names of all "Legally qualified Medical Practitioners" as aforesaid to be registered to be published in the Government Gazette on or about the first day of January and the same is to be repeated annually for the information of Coroners, Magistrates & the Public.

An Act—9 Victoria No 12—extended the privilege to Licentiates of the Apothecaries Hall of Dublin, October 27, 1845.

The first Medical Board in New South Wales notified in the Government Gazette of 12 December 1838 in accordance with the *Medical Qualifications Act*:

The Deputy Inspector General of Hospitals (Dr. J. V. Thompson), President.

Dr. Doble, R.N.
Dr. Robertson.
Dr. Nicholson.
Dr. Wallace.

[1] An Act to define the qualifications of Medical Witnesses at Coroners' Inquests and Inquiries held before Justices of the Peace in the Colony of New South Wales, 12 October, 1838; from the original in the Mitchell Library, Sydney.

Correspondence.

ACUTE ANTERIOR POLIOMYELITIS: ITS EARLY DIAGNOSIS.

SIR: As a victim of the disease, I have been particularly interested in the diagnosis aspect discussed at the plenary session of the recent congress (as published in THE MEDICAL JOURNAL OF AUSTRALIA, October 4, and in the symposium by J. Macnamara, S. Williams *et alii* recently in the same journal).

In an effort to diagnose this complaint early, and perhaps in time to prevent paralysis, might I suggest that during an epidemic, publicity be given to the following: (i) unexplained loss of appetite or "enjoyment for food"; (ii) motions looser than normal; (iii) increased fatigability and a general "achiness"; (iv) inability to coordinate eye, nerve and muscle—for example, in my case, after several holes, almost complete inability to hit a golf ball; (v) hyperæsthesias and paræsthesias.

These symptoms, in my case, were in evidence in an increasing degree for some weeks before paralysis. Point (v) became evident only five days before onset of paralysis. There was no pyrexia or headache until twelve hours before the onset of paralysis, and up to that time I was still practising. A method of diagnosis during these weeks may have left a different picture.

Also, in passing, might I refer to Dr. L. Hugh's remarks at the Congress. Towards the end of the recent poliomyelitis epidemic one of my partners had a frank Bornholm's, followed by signs most suggestive of a mild encephalitis with a negative lumbar puncture. This illness was followed by an orchitis. Five days later his wife had a frank meningoencephalitis, but with no definite paralysis, although cerebrospinal fluid findings were indistinguishable from poliomyelitis. Presumably, it was Coxsackie, and she recovered in two weeks. At about the same time both children of the family had ulcerated throats of a type described as being due to the Coxsackie virus.

Yours, etc.,
A. M. MACINTOSH.

Gerrale Street,
Cronulla,
New South Wales.
October 14, 1952.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, promotions *et cetera* are promulgated in the *Commonwealth of Australia Gazette*, Number 65, of September 25, 1952.

AUSTRALIAN MILITARY FORCES.

Royal Australian Army Medical Corps (Medical).

The following officers relinquish the provisional rank of Captain and are transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (1st Military District) in the rank of Honorary Captain, 28th July, 1952: QX700164 L. S. Stark and QX700165 A. R. Trist.

NX700381 Captain W. K. A. Paver is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District), 22nd July, 1952.

The following officers relinquish the provisional rank of Captain and are transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District) in the honorary rank of Captain: NX700382 I. Maxwell, 22nd July, 1952, and NX700380 K. M. Doust, 1st August, 1952.

The following officers relinquish the provisional rank of Captain and are transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (3rd Military District) in the honorary rank of Captain: VX700288 K. N. McNicol and VX700289 J. R. Officer, 2nd August, 1952, and VX700287 A. M. Dillon, 29th July, 1952.

To be Captain and Temporary Major, 23rd July, 1952.—VX700294 John Turner Hueston.

To be Captain (provisionally), 21st July, 1952.—NX700385 Joseph Ivan Davis.

Citizen Military Forces.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical): To be Temporary Major, 17th July, 1952.—2/79012 Captain G. Clifton-Smith.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/123264 Captain B. Clerehan is seconded whilst undergoing post-graduate studies in the United Kingdom, 1st April, 1952. 3/101817 Honorary Captain G. A. Rutherford is appointed from the Reserve of Officers, and to be Captain (provisionally), 19th June, 1952.

Central Command: Fourth Military District.

Royal Australian Army Medical Corps (Medical).—4/32019 Captain R. Barnes is appointed from the Reserve of Officers, 11th July, 1952.

Western Command: Fifth Military District.

Royal Australian Army Medical Corps (Medical): To be Temporary Major, 21st August, 1952.—5/26429 Captain (provisionally) K. W. H. Harris.

Tasmania Command: Sixth Military District.

Royal Australian Army Medical Corps (Medical): To be Captain (provisionally), 11th August, 1952.—6/15251 John Cairns Suetonius Officer.

Reserve Citizen Military Force.

Royal Australian Army Medical Corps (Medical).

1st Military District: To be Honorary Captains, 4th August, 1952.—Edward Franz Reye, John Francis Hennessey and Keith Ferguson Brady.

4th Military District: To be Honorary Captain, 4th August, 1952.—Ross Charles McKinnon.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

Desmond Boyd Goddard Bailie (039232) is appointed to a short-service commission on probation for a period of twelve months, 28th July, 1952, with the rank of Flight Lieutenant.

The probationary appointment of the following officers is confirmed: Squadron Leader R. D. G. Vann (024187), Flight Lieutenants D. J. Hannan (012786), F. I. Walke (012787).

The resignation of Flight Lieutenant D. H. Prentice (051149) is accepted, 19th July, 1952.

Active Citizen Air Force.

Medical Branch (Number 23 (City of Brisbane) Squadron).

James Taylor Duhig (015014) is appointed to a commission, 22nd July, 1952, with the rank of Flight Lieutenant.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course at Cooma.

THE Post-Graduate Committee in Medicine, in conjunction with the Far South Coast and Tablelands Medical Association, will hold a week-end course at Cooma on Saturday and Sunday, November 15 and 16, 1952. The programme will be as follows:

Saturday, November 15, at the R.S.L. Hall, Cooma: 2 p.m., registration; 2.15 p.m., "Recent Advances in Cardiology of Practical Importance", Dr. J. Kempson Maddox; 3.45 p.m., "Treatment of Insomnia in General Practice", Dr. David Ross.

Sunday, November 16, at the R.S.L. Hall, Cooma: 10 a.m., "High Blood Pressure—Medical Treatment", Dr. J. Kempson Maddox; "High Blood Pressure—Psychosomatic Aspects", Dr. David Ross; 2 p.m., "Duodenal Ulcer—Medical Aspects", Dr. J. Kempson Maddox; "Duodenal Ulcer—Psychosomatic Aspects", Dr. David Ross.

Fee for attendance at the course will be £3 3s. Those wishing to attend are requested to notify Dr. W. P. H. Dakin, Honorary Secretary, Far South Coast and Tablelands Medical Association, Sharp Street, Cooma, as soon as possible.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

THE Melbourne Permanent Post-Graduate Committee has pleasure in announcing that arrangements have been made with Dr. Denis Brinton, neurologist at Saint Mary's Hospital, London, and physician at the National Hospital for Nervous Diseases, Queen Square, London, to conduct a short course in neurology. This will comprise one lecture on "Effects of Cervical Spondylarthrosis on the Spinal Cord" to be given on Wednesday, November 19, 1952, at 8.15 p.m. in the Medical Society Hall, 426 Albert Street, East Melbourne, and two demonstrations in clinical neurology to be held in the Royal Melbourne Hospital Lecture Theatre from 1 to 2 p.m. on Monday, November 10, and Monday, November 17, 1952. By courtesy of the Royal Melbourne Hospital a buffet lunch will be provided from 12.30 to 1 p.m. on those days.

In order to facilitate arrangements, those desirous of attending are particularly requested to send their application beforehand, together with a cheque, to the Secretary, 394 Albert Street, East Melbourne. The fee will be £1 1s. for the course and 10s. 6d. each for the lecture and each demonstration. Resident medical officers will be admitted free of charge.

Obituary.

JOYCE SELDON STOBO.

WE regret to announce the death of Dr. Joyce Seldon Stobo, which occurred on October 19, 1952, at Brisbane.

LAURA MARGARET HOPE.

WE regret to announce the death of Dr. Laura Margaret Hope, which occurred on September 14, 1952, at Adelaide.

Notice.

FAIRFAX READING MEMORIAL PRIZE.

THE Committee of Management of the Dental Alumni Society's Fairfax Reading Memorial Prize has awarded this prize for 1952 to Dr. N. E. Goldsworthy. The prize is awarded

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED SEPTEMBER 27, 1952.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.[2]	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	1	..	..	..	..	..	..	1
Amœbiasis	..	4(1)	1(1)	..	..	..	..	..	5
Ancylostomiasis	..	..	2	..	..	..	1	..	3
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1(1)	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	2(1)	..	..	..	..	..	..	2
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	..	..	..	..	4	..	..	4
Diphtheria	4(1)	5(5)	10(5)	2(1)	..	1(1)	..	..	22
Dysentery (Bacillary)	..	3(3)	2(2)	..	1	..	..	..	6
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	..	..	..	..	10(8)	..	..	..	10
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	1(1)	..	..	..	..	..	1
Meningococcal Infection	7(5)	1(1)	1	..	1(1)	3	..	..	13
Ophthalmia	..	..	..	..	6	..	..	..	6
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	2(1)	8(2)	1(1)	13(11)	..	2(2)	..	..	26
Puerperal Fever	..	..	2(1)	..	..	..	..	..	2
Rubella	..	49(26)	..	..	17(1)	..	..	1	67
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	18(13)	39(23)	6(5)	4(4)	4(3)	7	..	1	79
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	23(20)	21(14)	14(13)	3(2)	12(7)	3	1	1	78
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	1	..	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 4, 1952.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.[2]	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1(1)	2	..	..	..	..	..	..	3
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	1	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	1(1)	..	8(7)	..	..	..	..	..	9
Diphtheria	3	1(1)	6(5)	..	1	..	..	..	11
Dysentery (Bacillary)	..	..	..	..	1(1)	..	..	..	1
Encephalitis	..	1(1)	..	3	..	..	..	..	4
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	..	7	..	..	14(10)	..	..	..	21
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	1	1	..	2	..	..	..	4
Leptospirosis	..	..	3	..	..	..	..	..	3
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	4(3)	5(5)	1(1)	1	2(2)	..	..	..	13
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	4(1)	5(5)	3	9(8)	..	..	..	..	21
Puerperal Fever	..	..	..	..	1	..	..	..	1
Rubella	..	41(30)	..	..	6(5)	..	..	3	50
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	20(11)	31(23)	10(10)	3(2)	5(5)	..	..	..	69
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	1	..	..	..	1
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	46(41)	40(31)	18(13)	6(3)	14(11)	..	2	..	126
Typhoid Fever	1(1)	..	..	..	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	1(1)	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

biennially and has been awarded this year to Dr. Goldsworthy for his outstanding services in the development of research and teaching in dental pathology, and for services to dental research generally.

THE OFFICIAL HISTORY OF AUSTRALIA'S PART IN THE WAR OF 1939–1945.

THE first volume of the medical series of the Australian War History by Dr. Allan S. Walker is to be published shortly, before the end of this year. It deals with the clinical medical and surgical problems encountered by the members of the medical services. The second volume of the medical series gives an account of medical matters in Australia and overseas up to the return of the Australian Imperial Force from the Middle East, and describes the experiences of the Eighth Division in Malaya in training, in action and in captivity. It will be published early next year.

The first volume of the military series, "To Benghazi", by Gavin Long, will be published before the end of this year, and it is hoped that the first volume of the civil series, "Government and the People, 1939-1941", by Paul Hasluck, will also appear about the end of the year.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additional qualifications have been registered: Kerkenezov, Nicholas (M.B., B.S., 1943, Univ. Sydney), Dip. Ophth., R.C.S. (England), 1951, R.C.P. (London), Dip. Ophth. Medicine and Surgery, R.C.S. (Ireland), R.C.P. (Ireland), 1951; Furber, Thomas R. (M.B., B.S., 1944, Univ. Sydney), M.S., 1952 (Univ. Sydney); Craven, Richard (M.B., B.S., 1943, Univ. Sydney), F.R.C.S. (Edinburgh), 1951.

QUEENSLAND.

THE following have been registered, pursuant to the provisions of *The Medical Acts*, 1939-1948, as duly qualified medical practitioners:

Foley, John, M.R.C.S. (England), 1950, L.R.C.P. (London), 1950.

Salvaris, Alexander, M.B., B.S., 1951 (Univ. Melbourne).

The following additional qualifications have been registered: Douglas, Robert Andrew, M.R.A.C.P., 1950, M.R.C.P. (London), 1952; Wilson, Harry Gilmore, M.R.A.C.P., 1947, M.R.C.P. (London), 1951; Wilkinson, Cyril Ignatius, M.S. (Univ. Queensland), 1952.

TASMANIA.

THE following have been registered, pursuant to the provisions of the *Medical Act*, 1918, as duly qualified medical practitioners:

Crawford, Matthew Scott, M.B., Ch.B., 1945 (Univ. Glasgow).

Clarke, Paul Stephen, M.B., B.S., 1948 (Univ. London).

Nominations and Elections.

THE undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Guy, Keith Butler, M.B., B.S., 1952 (Univ. Sydney); Jones, Robert Francis Clifford, M.B., B.S., 1952 (Univ. Sydney); Lush, Paul Joseph, M.B., B.S., 1952 (Univ. Sydney); Wells, John Graham, M.B., B.S., 1952 (Univ. Sydney); Carter, Leonard, M.B., B.S., 1945, D.P.H., 1947 (Univ. Sydney); Grieve, Neil McLean, M.B., B.S., 1951 (Univ. Sydney); Penna, Teresio, M.B., B.S., 1951 (Univ. Sydney); Thomas, Barrie Adrian, M.B., B.S., 1950 (Univ. Sydney); Ament, Leon, registered in accordance with the *Medical Practitioners Act*, 1938-1950, Section 17 (1) (*c*).

Diary for the Month.

Nov. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Nov. 5.—Western Australian Branch, B.M.A.: Council Meeting.
Nov. 6.—South Australian Branch, B.M.A.: Council Meeting.
Nov. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 17.—Victorian Branch, B.M.A.: Finance Subcommittee.
Nov. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 19.—Western Australian Branch, B.M.A.: General Meeting.
Nov. 20.—Victorian Branch, B.M.A.: Executive Committee.
Nov. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 25.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 26.—Victorian Branch, B.M.A.: Council Meeting.
Nov. 27.—New South Wales Branch, B.M.A.: Branch Meeting.
Nov. 27.—South Australian Branch, B.M.A.: Scientific Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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